





TRANSACTIONS

OF THE

TWENTY-SEVENTH ANNUAL MEETING

OF THE

American
Academy of Ophthalmology
and Oto-Laryngology

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HELD AT

MINNEAPOLIS AND ST. PAUL, MINN.

SEPTEMBER 19-23, 1922

The Twenty-eighth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at Washington, D. C., Oct. 16-20, 1923. The attention of the members is called to the ruling of the Council, that when a paper published in the Transactions is illustrated, the author must pay the total cost of all colored plates, and one-half of the cost of all other cuts. The Editor will furnish an estimate of the cost of such cuts and plates.

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ADDRESS OF WELCOME.

DR. LOTUS D. COFFMAN, PRESIDENT UNIVERSITY OF MINNESOTA.

MINNEAPOLIS, MINN.

A few months ago, when I was riding across the State of Nebraska, I read an editorial in an Omaha paper on Professor Mikelson's invention for the measurement of stars. The writer of this editorial called particular attention to the years of effort that Professor Mikelson had spent in devising an ingenious piece of apparatus to measure the diameter of stars, and that he had found that there were some stars that were a million times larger than the largest stars ever measured. Then the writer of this editorial said: "But we cannot see any connection between measuring the diameter of stars and the price of potatoes, or the price of corn, or the rent one has to pay."

The editorial really described two very radically opposed states of mind—one represented by those who believe that there is still some virtue to be found in a man who is willing to spend years in his laboratory poring over books and manuscripts and microscopes to discover new knowledge; and the other represented by those who believe that practically everything that we do should be directed to something that is immediately useful.

We have had this brought home to us recently in rather dramatic form, here in the Northwest. For a good many years, this section of country has been recognized as a great wheat belt. But areas that once produced large quantities of wheat now produce almost no wheat. Some few years ago, some of the scientists of this institution, who had been making investigations, declared that this region would never again be the great wheat producing region until the farmers, the wheat growers, the millers, everyone interested in the production of wheat took steps to eradicate the domestic barberry. The idea was scoffed at. The people said that wheat could grow wherever proper atmospheric and climatic conditions existed.

Recently I attended a meeting, where one of these young scientists spoke. He traced the barberry spores from the barberry stage through the grass stage, showed how they destroyed wheat and oats, barley and rye, and nearly forty kinds of grasses. The story he related sounded almost like a romance. When he finished it was clear that there was only one thing to do—either to

stop raising wheat or to break the chain of this barberry spore, because after the spore has produced upon the barberry, it will not reproduce upon the barberry again until it passes through the grass stage; after it has been generated upon grass, it will not reproduce upon grass until it passes through the barberry stage. Somewhere the chain must be broken, or we must quit raising wheat. Those who were present recognized the truth of his statements and took steps at once to eradicate the barberry.

Everywhere we find these two classes of minds—one represented by those who think that progress is to be made in the long run by the scientist who is willing to spend years in the study of some more or less remote fact, whose truth he does not understand or appreciate at the time; and the other represented by those who believe in practical, useful work, whose results can be appreciated at the time. I remember a number of years ago reading of the work of Pasteur, who had spent years in his laboratory hard at work, and who finally said he had found a cure for anthrax. He was invited to go to England by a group of livestock men, who said they would like to see his theories put to the test. He took a group of sheep and vaccinated part of them, but did not vaccinate the others. Then he went away, saying "When I return after a certain period, all the sheep in this flock will be dead, while those in this flock will be alive and well." The livestock men scoffed at him. They could not see how a virus of that sort could do what he claimed. When he came back, he found a group of men gathered about one of the flocks. Upon examination it showed that all of the sheep in the flock were dead, except three, and they were dying. His theories were put to the test and they stood the test, because of the careful scientific work he had done in his laboratory.

Now, gentlemen, we need both kinds of men. Progress in science can only be made by men who are willing to pay the price in terms of effort, in terms of years of devotion to a particular kind of study to discover new knowledge. I for one am quite willing to lift my hat to the quiet, bespectacled professor, who is willing to devote himself to the discovery of new knowledge. On the other hand, progress is to be made also by those who think in terms of results, who see things working out in some form of human activity. This University stands for both; it encourages both kinds of work. For that reason we gladly welcome your group, not merely because you represent the best in medical science, but because you encourage and appreciate the delver after truth and the searcher after knowledge.

If there is anything the University of Minnesota can do to make your stay here pleasant and profitable, we shall be pleased to do it. I am glad personally to extend you the welcome of the Board of Regents and the Faculty. Our services will be at your command. All you have to do is to ask for them, and you will find us willing and ready to respond.

ADDRESS OF WELCOME.

E. P. LYON, PH.D., DEAN OF THE MEDICAL SCHOOL, UNIVERSITY
OF MINNESOTA.

MINNEAPOLIS, MINN.

Mr. President, Members of the Academy: I consider this occasion unique in the history of the Medical School of the University of Minnesota; therefore it is with more than perfunctory pleasure that I welcome you here. It is a matter of some regret that your meetings will not be held in one of the buildings of the Medical School. On the other hand, we want you to see this building, which is the center of the life of the men students of the University in their social relations, and I hope also that none of you will go away without visiting the Medical School.

Our University is young; it is just past fifty years, and our Medical School will celebrate its thirty-fifth birthday next winter. So we are really a young institution. The scaffolding you see on the buildings here is symbolic alike of the external and internal life of the institution. We are in a growing state, and I hope when you see our Medical School, it will be eyes which not only see the present, but look through spectacles of imagination and see what the future has in store for us. You will find that we have an excellent beginning of our hospital system—about 200 beds—and fine buildings for scientific laboratories. We have a hospital plan of which we are proud, and hope some day to stand among the best in the country. This hospital system has been planned by Mr. Cass Gilbert, and we hope some day to have 600 beds instead of 200. We hope also that you will visit in your investigation the Todd Memorial Hospital for the Eye, Ear, Nose and Throat. (Applause.) This hospital was planned by your colleague, Doctor Frank Todd, who left among his papers memoranda covering the plans for its erection and his views as to what kind of work it should accomplish. From Mrs. Todd and friends of Doctor Todd, and through an appropriation made by the Board of Regents, this hospital will be realized within the next two years.

My own field of work has been in the fundamental lines of physiology, and you know that among biologists and evolutionists the question often arises, as to how it happened that all of the major senses are developed so near together at the head end of

the animal. The answer usually given by the evolutionist is that the ancestor of man being on his belly and going with the anterior end in advance, developed his senses there as a means of avoiding enemies and of making his way through life. I was thinking last night what a fine thing this is from the standpoint of the specialists in eye, ear, nose and throat. Suppose man's eye had been developed at the end of a long appendage remote from the digestive tract, like the star-fish, you men could not get together for the purpose for which you are here assembled. Or it might have been possible, and of some advantage, as I sometimes point out to my classes in physiology, if the openings to the respiratory tract had been quite apart from those to the digestive tract, as in the insects. Think of the complicated reflexes which would not have been developed, and think of the peculiar pathologic conditions which we might have avoided if that had been the case. But in that case, the associated specialties would not have been possible. I make these facetious remarks with the hope that your meetings may be as close and harmonious as the processes of evolution in the development of these senses and their anatomic structures with which you have to deal, and I hope that both from a scientific and social standpoint you will find your stay in Minneapolis profitable and pleasant.

PRESIDENT'S ADDRESS.

UNDERGRADUATE INSTRUCTION IN THE SPECIALTIES.

WALTER R. PARKER, B.S., M.D.

DETROIT, MICHIGAN.

I wish to take advantage of this, my first opportunity, to express to you my sincere appreciation of the honor you have conferred upon me, by electing me the presiding officer of the American Academy of Ophthalmology and Oto-Laryngology. It is a distinction I shall always regard as one of the greatest honors of my life.

Last year was a memorable one in the annals of the Academy, owing to its entrance into the field of education, which marked a distinct epoch in its history. The results of the work of the Sections on Instruction and Pathology were most gratifying, not only through the excellent program offered by the Section on Instruction, but also by the arrangement made by the Section on Pathology with the Surgeon General of the Army, whereby through the Army Medical Museum, the pathologic specimens collected, classified and interpreted, will be available for study. The congratulations and appreciation of the academy are due the members of these Sections for all they have accomplished, and they should receive the hearty cooperation of each Fellow.

In our enthusiasm for the expansion program, it is well not to forget that all the activities of the Academy are secondary to the regular program that is offered each year. The major portion of the work incident to the preparation of this program falls on the Secretary, and I wish to express my great appreciation of the splendid results of his untiring efforts in this connection.

In view of the entrance of the Academy into the field of medical instruction, it seems proper to bring before you any aspect of the subject that may be of immediate interest to our branch of the profession. Great advance has been made during the past few years in the character of Postgraduate instruction in Ophthalmology and Oto-Laryngology, and in several places instruction is now very well given, and inadequate and incomplete courses are being eliminated. The

problem which I wish to consider in particular has to do with the importance of undergraduate instruction in Ophthalmology and Oto-Laryngology, although what I have to say applies equally to the status of the branches of Medicine and Surgery that are classified as specialties.

Before taking up the problem of undergraduate teaching in detail, I shall give a short review of the development of the methods of medical instruction in this country. Not many years ago, the medical schools were graded according to the excellence of the clinical work given, the laboratory side being secondary in this classification. Later, as the laboratories were developed and the inaccuracies of many of the clinical methods of diagnosis became apparent, the standard of classification of the medical schools changed, and those that had the best laboratory equipment in apparatus and personnel were given first place. The advancement in medical training on the scientific side was marvelous during this second period. As a result, the tendency was to develop workers in clinical laboratories, while the number of graduates who were interested in the clinical care of patients, grew relatively less.

In the third phase of this development, a correlation of the clinical and laboratory branches of clinical instruction was effected, that has resulted in our present technic of medical teaching. In the inauguration of this system, attention was concentrated on the so-called clinical years of instruction—that is the last two years of the medical course. The methods of teaching in the preclinical—or first two years of the course—have undergone comparatively slight change, so far as the clinical aspect of the work is concerned.

We are now entering the fourth phase of the evolution in medical teaching, and already a few schools are introducing the idea, that the work during the first two years should be given a more clinical interpretation. In so doing, it is not the importance of a thorough grounding in the subjects taught in the preclinical years that is being questioned, but the advisability of bringing out, as far as possible, the clinical significance of the instruction given during this period. In the event of this change, what is now known as the preclinical course would become in a degree an integral part of the practical medical training.

Having briefly outlined the changes that have taken place in the program of general medical training, I will next con-

sider the question of the importance of courses in the specialties in the undergraduate curriculum.

With the phenomenal advance made in all branches of medicine and surgery, not only have the fully established specialties been placed on a firmer basis, but others also have been developed. I believe it can be said, without exaggeration, that one of the dominant factors in the progress of medical science has been the influence of the work accomplished by the so-called specialists. In spite, however, of all that has been contributed by the special branches of medicine and surgery, it has been proposed to abolish courses in the specialties from the undergraduate curriculum of medical schools and colleges. To be more explicit, at the last meeting of the American Medical Association, the Committee on Education of the House of Delegates made the following recommendation, which was adopted:

"It is urged that medical schools so revise their curriculums as to provide a thorough training for the general practice of medicine, leaving courses in the specialties for the graduate medical school."

I entirely agree with those who contend that the undergraduate teaching of the specialties should be limited to the requirements necessary for a thorough general medical training, and that the development of specialists should be left for postgraduate work. There is, however, a great difference between the training of specialists, and the teaching of the essentials of the special branches of medicine and surgery to the undergraduate. Much confusion has been shown in some of the discussions on this point. I recognize the fact that the present curriculum is overcrowded, and that a proper balance should be maintained between the various subjects taught, but some time must be provided for instruction in the special branches of medicine and surgery, if students are to be given a thorough training for the general practice of medicine.

What should be the standard of requirements for the undergraduate medical student? A thorough knowledge of the fundamentals is of course essential and does not enter into this discussion, which applies only to the subject of the clinical instruction. Practically, it would appear proper to acquaint the student of medicine with the methods of examination, and with the management and treatment of those diseased conditions with which he will come in contact as a general practitioner.

That relative values are sometimes lost sight of is well shown in the following incident: In a recent state board examination, the Chairman discoursed on the advisability of the applicants refraining from entering the specialties, and confining themselves to general practice. This may have been an entirely proper admonition, but hardly consistent in view of the question asked in the examination that followed, namely, "Give in detail, a description of the technic of a hip joint amputation." Applying the test of practice to this question, how often would a hip joint amputation be performed by a general practitioner as compared to the number of times he would be required to recognize and treat cases of acute otitis media, infected tonsils, sinusitis or mastoiditis? Or, again, what would be the relative importance of this knowledge of general surgery to the general practitioner, as compared to his ability to appreciate the possible clinical manifestations of errors of refraction, or to recognize cases of corneal ulcer, iritis or glaucoma? Yet, it is recommended that courses in specialties be left for the graduate medical school. I admit that these are extreme examples, but they serve to illustrate in a striking way the possible end results of the proposed changes. While these changes affect primarily the undergraduate curriculum, it is apparent that they vitally concern the advancement of medicine and surgery, by inhibiting the development of the special branches.

It is difficult to escape the thought that the originators of these changes were influenced, not so much by a desire to do away with many of the subjects taught, as to exercise control over them by introducing them into major services. In other words, they run the risk of permitting their interest in the administration of the various departments to overshadow their concern for the quality of the work given, and for the methods of educational technic. To my mind, there is no question that it would be infinitely better to maintain a proper relation between strong departments, than to render ineffectual the special branches of medicine and surgery by destroying their independence.

Stripped of all its collateral complications, the matter is reduced to the consideration of two questions, first whether or not Ophthalmology and Oto-Laryngology should be administered as separate departments, or as sections in the department of Surgery; and second, whether the departments as now constituted could give more practical courses.

In answer to the first question, it seems reasonable that by giving each department full opportunity to develop, better men will be held as heads, and consequently a higher quality of instruction secured, than would obtain if the work were delegated to an assistant in the department of Surgery. While some of the technical subjects can be taught just as well, perhaps better by assistants, the broad aspect of each specialty in its relation to a general medical training can be adequately presented only by one of wide clinical experience in that specialty. Besides contributing to the undergraduate program, a properly organized special department should be so administered as to encourage the spirit of research in the members of its staff and to serve, through scientific contributions, the institution of which it is an integral part. To accomplish these results, each department should enjoy complete autonomy. Authority to administer the affairs of a department is quite as essential to its development as the capacity for assuming responsibility for the character of the courses given.

The second question, whether or not the most practical course possible is given in every instance, must, I believe, in all fairness be answered in the negative. There is great variation in different institutions in the method of teaching the specialties, and some standardization is most desirable. Just how this is to be accomplished is not clear, but I am convinced that the remedy does not lie in the subordination of the special departments, but rather in establishing a proper working relation between the existing departments, each one retaining its independence and being administered by an experienced clinician.

All special departments in a medical curriculum have a twofold function to perform—

1. To establish the relation between the work given by the special department and that required in the general medical training.
2. To familiarize the student with the methods of diagnosis and treatment of the more common diseases seen clinically in each department.

The first duty is to teach the interpretations of findings, rather than the management of the specific lesion. The knowledge that an infected tonsil may give rise to a multiple neuritis, uveitis, or a gastrointestinal reflex disturbance, is more important to the student of medicine than a demonstra-

tion of the operation of tonsillectomy. Again, the knowledge that the sudden onset of double vision may be associated with tabes, multiple sclerosis, general paralysis, or be a result of a peripheral neuritis, is much more important to the student, and the patient, than a detailed description of the methods employed in the differentiation of the particular muscle affected. The latter of course is essential, but comes properly in the province of postgraduate instruction. Many similar examples could be given.

Those who are devoting their major attention to the branches of medicine and surgery which by universal consent are classified as specialties, are often accused of a narrow viewpoint. But in point of fact, is their view any more narrow, or even as narrow, as that which is being promulgated by those who are concerned with general surgery and medicine, when they fail to appreciate the relative importance which those so-called specialties must maintain in the educational program of the undergraduate, in preparation for his equipment for future practice?

No suggestions will be offered as to the details of a schedule for undergraduate courses in Ophthalmology and Otolaryngology, my object in presenting this matter being to emphasize the importance of these subjects in the curriculum of a properly arranged medical course, and to call attention to the proposed radical changes, the adoption of which would, in my judgment, be a step backward in the science of medical teaching.

DISCUSSION.

DR. CASSIUS D. WESTCOTT, Chicago: I think we have heard a very interesting address, which contains some suggestions and presents some facts upon which we may wish to act later. I therefore move, Mr. Chairman, that a committee be appointed to consider carefully the President's Address, and present some suggestions for us to act upon in this body.

Motion seconded by DR. J. A. STUCKY, and carried.

CASE TAKING AND CASE RECORDS.

EDWARD JACKSON, M.D.

DENVER, COLO.

The most general characteristic of medical cases is, that they are all different—there are no two alike. Assume a case of refractive anomaly; all cases have some refractive anomaly; one-half of our patients come because of errors of refraction. But this patient has myopia; only one in twenty has myopia. He has astigmatism. The majority of patients have astigmatism; but in this case it is confined to one eye and is at an “off” axis. Next year you find the astigmatism has changed in direction and increased; there is a loss of visual acuity and you discover a marked conical cornea. When you go back over the patient’s general history, you find that preceding the change of astigmatism there were symptoms of duodenal ulcer. In over 40 years I have seen but one case of conical cornea with that history, although in nearly all cases of conical cornea, a history of some impairment of health probably underlying it could be obtained. Has any one here seen a case with such a history? If so, probably a comparison of cases would quickly show a dozen points of difference, and among these points of difference might lie the most significant facts regarding either of the cases. These are a few of the possible variations in an “ordinary refraction case.”

The pathologic variables are so numerous, that a mathematic calculation would show that no two cases encountered in a lifetime could be expected to be alike. We see scores of faces every day, yet throughout a long life never two just alike, and rarely two with a puzzling resemblance to one another. When we think two faces are alike, it is because we have not observed them closely enough to note the differences, and the same is true of cases. If at first glance two cases seem quite similar, the differences quickly appear with closer study. If at a certain time there is a close resemblance, the histories of them are found to differ, or their clinical courses quickly diverge.

If we “follow” the case, we will be led, somewhere, in a different direction from that taken by any similar case we

have ever seen. Its outcome will be different; either its response to treatment will vary from what previous experience would indicate, or its rate of progress, or the general outcome, or the sequels or complications that arise, will stamp each case as different from any other. One of the first and most important lessons the medical student has to learn is, that the case he sees differs from any case he has read or heard of, does not conform exactly to any one type, does not lie wholly within the limits marked out in the textbooks for any one form of disease.

Every case study is a bit of original investigation. If it notes only what was significant in some other case, something important will be overlooked. If it considers everything that was important in some other case, it will never reach any intelligent grasp of the situation; but will result in an irrelevant mass of details and useless confusion, which will hinder or prevent solution of the real problems that should have claimed attention.

If you go to see a patient, you do not traverse every possible street and look at every house number to find the one you seek. You take the information, or the clues you have, and from your knowledge of the city proceed along what seems the shortest, easiest way to your goal. Even the hospital you visit every day is not always reached by the same route. Knowing in a general way where it is, and the chief landmarks of your city, you come to it along different streets each day of the week, taking the most direct way from where you happen to be when you turn to go to it.

So it is with cases. The symptom first seen or the patient's complaint starts the quest for conditions present, probable causes and measures for relief. When the prominent features of the case have been recognized, when the general scope of the study has been indicated, each part of the ground included must be carefully worked over to make sure that nothing important has been overlooked. But such a minute survey is confined to things germane to the case; it never can cover all the field of medicine or all of a field of special practice.

The study of a science or an art may include attention to every possible detail of every part of the subject, like the mapping of a county or the taking of a census. The study of a case should be more like going from one place in the city to another; concerned only with the direct route, and the possible side issues into which it is easy to be led, and

which must be avoided if the destination is to be reached in the shortest time. When we get to the right street and the right side of it, we can begin to look for the number of the house. The quicker we get into the right locality, the more time there will be left for minute investigation, where minute investigation is needed.

ROUTINE EXAMINATIONS.

Routine examination, supposed to cover all the domain of medicine, or all of any special branch of medicine, or everything with regard to a particular class of diseases, or all that might relate to one particular disease, has no place in case study—would embarrass and defeat case study. Our minds are not infinite; we cannot grasp or coordinate all possible facts or all discoverable conditions at once. The good diagnostician is like the keen scented hound, that catches the scent he is to follow and sticks to it, without concerning himself with the cross trails and the smell of every thing he may pass.

The diagnostician arrives at his diagnosis as the hound arrives at his prey, by single minded, direct pursuit. He might have accumulated a whole encyclopedia of information along the path, but it would not have helped him to his purpose. It would necessarily have delayed him and very likely have turned him entirely from the true course, or loaded him with detail that rendered him powerless to move any further in the quest.

No routine examination or combination of examinations gives the mastery of a case. Successful case taking does not consist in "thorough examination" of the patient for symptoms and conditions that he does not have; but in eliciting those that he does have; in determining the absence of others that might be expected to be associated with them. The system is like the following of a trail; sometimes getting off the right trail, but finding this out and getting back to it as soon as possible; and skill is shown by keeping to the right line of thought and investigation, and quickly getting back to it when a mistake has been made.

Routine examinations should be confined to a very few things that are significant in practically all cases; or which furnish data that will be most useful in estimating the progress of the case. The starting point is commonly the patient's statement, or complaint. This is our contact with the patient

and the opening of the trail to be followed. For eye diseases, the acuteness of vision should be taken in every case. It is more constantly suggestive as to the nature, course and progress of the case than any other one symptom. Inspection of the patient, more or less careful, can hardly be avoided, and may give the clew to a diagnosis before the patient can make any statement. Then, for all cases in which it can be made, the ophthalmoscopic examination should be resorted to. It tells so much at a glance and is so often valuable through furnishing a basis for estimating later the progress of the case, that it should be counted as part of the routine examination.

For each general class of conditions, certain routine tests should be made. For abnormalities of refraction or ocular movements, the near point of accommodation and muscle balance should be determined; for conjunctival disease the bacterial content of the conjunctiva. But the bulk of case taking is investigation to ascertain the presence, variety, stage, severity, special form and complications of some particular disease. The rehearsal of what such investigations include, covering all of ophthalmic and much of general diagnosis, is here impossible. The purpose is to show how the problems of case taking are to be attacked; along what general lines the investigator's knowledge of ophthalmology, general medicine, anatomy, physiology, physics, psychology, in brief the intellectual resources and armamentarium of the practitioner, are to be brought to bear on case taking.

CASE RECORDS.

A good case record is as individual as the case; as individual as the investigator. The characteristic of individuality, with a common general plan of arrangement, is about all that medical case records can properly have in common. This does not need to be enlarged upon; it follows from what has been pointed out of case taking. As cases are not duplicates one of another; proper records of them cannot be. Elaborate blanks for case records are chiefly waste paper and useless printing. Even if you have a line for "redness of the eyeball," that can be checked off to save the trouble of writing it, it does not tell the severity, the location, the hue or any significant fact about the redness. These must be written in, or the record is left very deficient. To provide for all possible variations of this kind, a score of lines would be required, scattering the record of the individual case, requiring search and reading

of a paragraph instead of a phrase whenever the record is to be consulted, for what might be recorded by three or four written words.

Elaborate blanks for case records are of some value, as showing what their authors thought the most important things to be noted. They may also serve as reminders of points to be looked for, should the user be inexperienced in diagnosis. But for the latter use they are a dangerous reliance. The suggestion of what to look for in any given case should come from what has already been learned of the case, not from a printed page largely occupied by matters not bearing on the particular case. When the case fails to suggest lines of inquiry to the surgeon, his usefulness as a diagnostician is not great; and to learn to rely on such extraneous aid, rather than on an alert well stored mind is a dangerous habit.

The schedules for systematic examination of the patient found in some excellent textbooks are intended to suggest to the student the scope and order of examination that he must learn to make, and not to save him from the need and effort of mental alertness, when he is actually engaged in case taking. It is essential to note: "This schedule of examination must be modified to suit individual cases." (de Schweinitz.) It is only a help to establish paths and habits of thought. Until one's individual methods of regarding and hunting down the case are developed, very little real help has been received from such a schedule.

If you use such a record blank printed in black, it is best to write your notes in red, so that the real facts in the case will stand out from the background of useless words that might fit other cases. But the most serviceable record blank will have few things printed on it. Only the blank page has the perfect elasticity that a record blank needs, to conform to the infinite variety of cases. The "paper work" of the late war illustrated how much time can be wasted in filling in details that might be significant in some other cases. Yet this might be excused in records of units supposed to be all alike, when it is inexcusable in supposed scientific records of pathologic conditions, that are always variant from those of any other case, and which are generally worth having because the conditions present did differ from those in other cases.

A useless waste of space and energy is often seen in the recording of negatives. Sometimes they are significant, as

showing that a diagnosis is reached or confirmed by exclusion, or that a test has been applied, or a symptom looked for. But, as they often appear in published case records, their most obvious purpose is to impress the reader with the up-to-date methods and thoroughness of the writer. If the case record is worth reading at all, it presents a fairly accurate picture of the pathologic condition that actually existed. It is no help to be reminded that there are other conditions that were not present. At least, negatives should be stated so as to involve the least possible waste of writing or reading, whether for private use of case records or in their publication. Like everything else in a case record, they should throw light on that particular case.

A passion for completeness sometimes leads to noting details that have no practical value. The unselective mind can easily produce case records that are wearisome and largely worthless. A certain ophthalmologist noted on his records of his ophthalmoscopic examinations the proportion of the horizontal and vertical diameters of the optic disc as 4-5, 9-10, etc. Such notations in the nature of the case could not be exact. The apparent relation varied with the real shape of the disc, its obliquity and the astigmatism of the eye. Only very rarely could such records have significance, in progressive myopia indicating increased obliquity through scleral bulging. Many times, moreover, the fixing attention on an unimportant detail must have caused more important appearances to have passed unnoticed. There is a real danger that habit and routine will hinder the noting of what is especially important in the individual case.

ABBREVIATIONS.

The mechanical labor and time consumed in the writing of case records is a serious tax on the time and energy of the busy practitioner. This is urged as a sufficient reason for not living up to the rules of the American College of Surgeons for case histories in hospital standardization. It will be a real gain if the labor is reduced to a minimum, compatible with the statement of all essential facts; and this is approached by noting only what is essential, with the words and symbols that require the least space and time, either to set down or read. The matter of abbreviations is so important that it is worthy of separate consideration, and is most important in connection with the taking and publishing of

case records. Every good abbreviation should be more generally used. The discussion of them, agreement as to which are best and their formal adoption in our Societies, would extend their benefit in case records.

It should be remembered that the benefit of using abbreviations is not simply in the time and labor saved in writing case records, and the economy of space in storing them. Where observations made at different times are to be read and compared, as the visual acuity or state of the muscle balance, good abbreviations not only save time and make comparison easier, but they also make it possible to get a more general and comprehensive grasp of the situation than would be possible without them.

INDEXING.

A library is a collection of books and other printed matter, arranged and catalogued. Without the arrangement and cataloguing it would be waste paper. Without some system of arrangement, case records are inaccessible and useless. Arrangement by writing on the successive pages of a book gives a chronologic order. Arrangement by the patient's name, or indexing by names the records in a book, is the convenient method for purposes of treating patients.

So long as one can remember his patients by name and the peculiarities of their cases, the arrangement or index by name will serve other purposes. But as time goes on and records accumulate, other kinds of indexes become necessary. An index of diagnoses, one of treatment, perhaps one of operations, are of value in utilizing the benefits of one's own experience. The card system of keeping records lends itself readily to multiple indexing, and makes it easy to throw any group of case histories together temporarily for purposes of special study. Such records may be given a chronologic order by giving numbers consecutively to each new case; or as is more convenient in practice, they may be arranged alphabetically by the patient's last name. In any case it is worth having all the indexes one will use frequently, but never more than will be kept well up to date.

EDUCATION IN CASE TAKING AND RECORDING.

Enough has been said to illustrate that the methods of taking and recording cases are of sufficient importance to justify their discussion in our Society meetings, and special training in their preparation for general medical and special

practice. In the main, training in this very important branch must be given apart from books, although Cabot's "Medical Case Teaching" is serving a very useful purpose, and there is room for a carefully prepared work on case teaching in our specialties.

The real mastery of this part of our art still will be gained only in active work, studying and recording cases, under one who has developed his own methods to high efficiency and is a good teacher. Every good clinical teacher is fitted for teaching case taking, at least by the example of his method of attacking case problems. If the teaching is to a group, small enough to permit of free questioning and discussion, and for the teacher to watch closely and criticize the system and methods of the student, the conditions for such training are most favorable. A round table discussion of this subject would be appropriate for courses of graduate education in connection with local societies devoted to ophthalmology or oto-laryngology.

DISCUSSION.

DR. ROBERT SCOTT LAMB, Washington, D. C.: It seems to me we would all have a little different mental attitude toward case taking, if we felt that coming behind us was someone who would check up our work. I feel we are a bit overconfident that we will hold our patients. We do not always do it. We are too careless, too well satisfied that the man who sent the patient to us gave us a good send off, when as a matter of fact it may have been only lukewarm. I believe if we felt that somebody was coming behind us to check our work, we would not only make fewer mistakes, but our records would be very much more satisfactory.

One of the most important things is to get the confidence of your patient, to be sure that you have his cooperation. This is especially true with children—encourage them, persuade them. After the complaint is made known by the patient, a few leading questions will bring out any further history in connection with the immediate condition. History taking is the most valuable work you do. In your future consideration of the case, you will find the time spent in history taking is time well spent. After a few words with the patient, you begin your examination of the eye with an examination of the patient's face—whether he is wearing glasses, and whether the glasses fit. Examine the eyelashes, the margins of the lids, the under surface of the upper lid, the exposed surface of the lower lid, by version, to see whether there is any follicular conjunctivitis or any accumulation of mucus. All these things can be done quickly. Then your patient is ready for examination of muscle balance, after which, vision and refraction. If you are satisfied that there is any need for field taking, you take the field. You take the pupillary reflexes, and then you are ready for the cycloplegic. That leads me to say that I do not believe we ought to allow any case to go out of the office—any of these refraction cases—without a thoroughgoing examination

of the background of the eye, and this cannot be made without thorough dilatation of the pupil.

Now you have made a fair start in getting the data on which to base your judgment of the conditions present, and what ought to be done. You have, in addition to the soil—which is the complaint and the findings—a subsoil which goes into the environmental conditions, the inherited conditions in the life of the patient—the temperamental makeup due to inheritance. The field here is unlimited; you can go as far as you like, and I think that some of us who go into that field are greatly interested. You will have cases where the thing that seems unimportant to the patient is the thing that is prolonging the corneal ulcer, or the persistent headache; and it may be that some of these things that are unimportant to the patient will be found to be definitely causative. If we go into this field, it should be very carefully and very thoroughly done, and I would recommend it to all who feel they can afford the time. I believe after a while you will learn short cuts to the keynote of the condition, and find the causative factors in connection with it. I am perfectly satisfied that you will never cure these “old rounder” headache cases unless you get down to the basic causative factors of the condition of which the patient complains.

DR. H. V. WÜRDEMAN, Seattle Wash.: As regards the material used in case taking, Doctor Jackson shows us that simplicity of records is a sign of sense. Up to a couple of decades ago, the paper work of the average medical man was limited to a pocketbook for calls, with hieroglyphic signs for his charges. The keeping of case histories simply was not done.

With better medical education, the dying off of the two year graduates, and the advent of specialization, physicians generally began to keep clinical records of some kind. But even now, as “we who fought the war” (mostly in the encampments) well know, the taking of a proper case history, and more particularly its preservation on paper, is not practiced in the general profession, however well we special surgeons keep them.

We first started out with books, inelastic, requiring repetition on different pages and in different books, which in turn were superseded by the loose leaf and card systems. For general medicine, those in use by the U. S. Army and Navy are incomparably the most exhaustive. Many of us remember Forms 55 b, c, d, etc., and all have had opportunity to fill out the newer standardized hospital sheets. These contain too many pages and too much matter extraneous to our specialties. So most of us have resorted to card systems, varying from simple, plain sheets to elaborately printed forms. Here a happy mean is best—a simple skeleton of printed matter to be filled in by writing or typing, in which normal conditions are checked and only abnormalities usually noted.

Our advice is to restrict paper work to the minimum consistent with clearness and comprehensiveness, so that another surgeon could take up the case with a sufficiency of knowledge from its inception and through its course.

Some of our consultants credit us with marvellous memories, because we recall patients and their previous history, but to tell the truth, most of us have everything written down on paper.

DR. J. M. BANISTER, Omaha, Nebraska: I think, in our eye records, we should not overlook the source of focal infection in the accessory sinuses of the nose, often the tonsils and teeth. I have seen papilledema get well from operating on an infected antrum, from the removal of a tooth; also ulceration of the cornea remain unimproved until infected tonsils were removed. Therefore I think, that in all of our ophthalmologic work, we should not forget the otologic and rhinologic side. I think those of us who are oculists only should have at hand a competent nose and throat man, to look into our cases of ocular inflammation. You remember Dr. de Schweinitz' most magnificent monograph, presented before the International Congress, on Chronic Uveitis and Similar Diseases resulting from Focal Infections. We should not forget these lessons which the great masters in our branch of medicine have taught us.

DR. WILLIAM C. BANE, Denver, Colorado: The valuable points brought out by Doctor Jackson give us some idea of the thoroughness of his method of case taking and entering records.

The habit which the author has of sketching or illustrating pathologic changes or anomalies, tends to closer observation and care in entering records. A very important point, too often neglected, is entering upon the records the changes which take place from time to time as the case progresses.

Often a picture of an eye is desired to add to the records. I have secured a very satisfactory photograph of an eye, by extending the bellows of the kodak and adding a + 3 or 4 diopter sphere, in a portrait attachment, in front of the lens. I have been able to shorten the focus to obtain the desired enlargement. It is necessary to get the exact focus. I use a Junior-A Eastman Kodak, pulling out the bellows to get a 3 foot focus from the back of the kodak. Then by using a + 4 diopter lens in the portrait attachment, I shorten the focus to $14\frac{7}{8}$ inches from the back of the kodak.

DR. GEORGE F. KEIPER, Lafayette, Indiana: The paper of Doctor Jackson is very apropos, because we should all aim to keep good records. In our hospital work at home, we make such endeavor. That it may be accomplished, it is necessary to have inspection of the records by a nonpartisan member of the staff, who will grade the records. At the monthly meeting of the staff, he reads the grades attained by each member the preceding month, so that the men strive to excel, and become very careful in the preparation of records.

Why do we take records? The ultimate purpose is to secure accurate diagnosis, and unless the record is accurately taken and recorded, we cannot attain that end.

As to the printed form, I must reluctantly disagree with Doctor Jackson. We all, I trust, have good memories, but we also have good forgetters, and unless we have before us all that we ought to look for, we are liable to miss an essential detail.

We are apparently discussing eye records, but the general principles laid down will apply equally to the ear, nose and throat men.

DR. ALBERT H. ANDREWS, Chicago: Every man will make the records which appeal to himself. My own preference is for a plain card. In my private work, I do not care for an elaborate printed record blank. But there is a matter which I think is of quite great importance, and

that is the making of diagrams. Suppose you have a foreign body in the cornea. If you make a circle for the cornea, another circle inside for the pupil, and mark the location of that foreign body, you may save yourself a little trouble if later an ulcer should develop in some other part of the cornea. I believe it is a good plan also to make diagrams in ear work and in nose work—for instance, in deflection of the septum. Two or three diagrams can be made a permanent record in the office. After the patient is operated upon, there is no way of knowing the condition that existed before the operation; but you can keep this information by a diagram, which can be made in one-fourth of the time it takes to describe it.

DR. VERNON A. CHAPMAN, Milwaukee, Wis.: Sometimes we overlook in these examinations the most important point. It is surprising, in my work with students on cases at the County Hospital, how many cases will be brought in with elaborate examination records, and yet if you say, "Doctor, how much does this man see?" you find the vision was not taken. It seems to me, in these cases, the vision is just as important as any other function of the body. So important is this, that it seems to me in every case the first thing to do is to take the vision of the right and left eye, with and without glasses, and record it. I believe this should be done before the eye is touched, before the lids are everted, because so often the patient says he could see better before the lid was everted.

DR. OLIVER TYDINGS, Chicago: I agree with Dr. Banister, that a man is sadly handicapped in doing only eye work. Men who have attained to the exalted position of Doctor Jackson will enlist the aid of men trained in nose and throat work; but younger men, however capable of making a diagnosis of anatomic structure involved, would fail to recognize the relation between those conditions and nose and throat pathology. The same is true of every man who enters this field through the gates of special training, without a broad experience in general medicine.

DR. EDWARD JACKSON, Denver, Colo. (closing): The discussion, I think, illustrates the breadth of the subject. Every speaker seemed to go outside the line, and yet he was strictly following paths that go out from the center established by the paper. The comparison of a large number of routine methods means improved methods of examination and of record, and when they get through discussing them, nine times out of ten the methods will be simpler than they were to start with. When a man brings his own printed blank, one that he probably thinks is a concrete and visible schedule of his methods of work, one that is worth more to him and his students than anything else, it means that in the working out of such a blank he has gotten an amount of training, that he would have been a long time in getting in any other way.

The breadth of the investigation that may be strictly applicable in case taking has been illustrated by two or three speakers. That could not be touched upon in the paper, except that case taking calls for the mental alertness, the energy, the whole knowledge and breadth of view of the investigator.

SOME POINTS OF TECHNIC IN A COMBINED INTRA- AND EXTRANASAL DACRYOCYSTOTOMY.

(LANTERN DEMONSTRATION.)

J. SHELDON CLARK, M.D.

FREEPORT, ILL.

Conservation of anatomic structure and continuance of function is the basic reason for all that has been said and written by those who have worked on the proposition of the cure of dacryocystitis by an intranasal, shortcircuit drainage of the lacrimal sac, proximal to the junction of the sac and the nasolacrimal duct.

As to the merits or demerits of other methods of treatment, I do not care to speak, since they are not my concern in this paper. That they are not all ideal in every situation of tear sac disease is evidenced by the fact, that so many and varied methods have been brought forward during the past ten or twelve years, each with its enthusiastic proponent.

By way of introduction, you who attended the meetings of this Society in 1914 at Boston, and in 1915 at Chicago, will recall my presentation of this subject. Particularly in 1915 did I offer you additions to the technic of the then little known intranasal operation of West. Particularly would I call to your attention the citation in the paper of 1915, of an experience with a case wherein I made a stab wound directly into the tear sac, in order to better orientate myself with the use of the probe. This was done on account of a closed lower canaliculus, which in turn was caused by previous faulty probings and subsequent infection of tissues, including corneal ulcer and loss of vision. Likewise, a nasal sac punch forceps was presented for punching out the sac.

As to the developmental stages of the anatomy of the nasolacrimal passageways, one can do no better than consult the monumental work of Dr. J. Parsons Schaeffer, (P. Blakiston's Son Co.) entitled, "The Nose, Paranasal Sinuses, Nasolacrimal Passageways and the Olfactory Organ in Man;" wherein he devotes chapter VII of that work to "The Nasolacrimal Passageways."

Of the variations and anomalies that may take place, a mention should be made of a few as cited by Schaeffer; such

as double and triple puncta and lacrimal ducts, absence of lacrimal duct and papilla, and lacrimal punctum represented by slit like furrow along the edge of the eyelid. Congenital fistula is seen, and of this I now have a case under observation, which is of the unilateral variety. Auxiliary buddings of the nasolacrimal duct occur at one stage of the human embryo and are very common. These buddings, in many instances, suffer early absorption. Schaeffer is convinced that many of the diverticulæ of the adult nasolacrimal duct have their origin in these embryonic lateral buddings. These diverticulæ are always connected with the main duct, and are lined by an epithelium similar to that of the nasolacrimal duct.

The fossa of the lacrimal sac varies greatly in its bony conformation. One should have this in mind when doing this intranasal surgery. The ventral portion of the fossa is formed by the nasal process of the superior maxillary bone, and the lacrimal bone forms the balance of the fossa. These bones likewise go to form the lacrimal groove, which in turn lodges the nasal duct. The proportion which the nasal process of the superior maxilla and the lacrimal bone take to form the fossa for the sac, varies in different skulls. It may be about equal or it may be of varying proportions. The density of the bone oftentimes determines the readiness with which one is able to remove the bone of the lacrimal fossa. There are the anterior and posterior lacrimal crests, and these limit the fossa ventralward and dorsalward. The sac in all instances should be outlined by these anatomic landmarks, and can be more readily done in the aged.

The nasolacrimal canal is usually directly continuous with the lacrimal sac. One must bear in mind its close relationship with other structures, such as the antrum, on whose nasal wall it forms a protuberance. The agger nasi cell, or anterior ethmoidal cell, may lie just anterior, posterior, or may overlap the region of the swelling of the sac floor on the lateral nasal wall, which we term the *torus lacrimonalis*. Again, the middle turbinal may overlap this region.

The outlet of the lacrimal sac is through the junction of the sac and duct. This is contained in a fixed bony framework. Within the bony portion of the canal, there are two layers of cylindric epithelial cells, which according to Parsons (Pathology of the Eye, Volume II, Page 750), are very high, being 35 to 50 microns. There is a definite basement membrane. Beneath the basement membrane is an adenoid layer.

The submucosa contains dense fibrous tissue, which is very vascular, especially on the part adjacent to bone. Therefore, by studying the pathologic anatomy of the nasolacrimal passageways, one can well see how it is that we are often confronted with diseased conditions of the lacrimal sac.

Lack of drainage is one of the strong factors in keeping up a diseased condition of the sac. We know that many points of secondary infection in the body are located in structures which are subject to periods of congestion and decongestion, such as the introitus of the esophagus, the cardiac and pyloric ends of the stomach, the biliary apparatus, the appendicocolic valve, etc. So it is with inflammations of the nasolacrimal passageways, and a very large percentage of the inflammatory areas are situated within the bony part of the nasolacrimal duct, and these in turn block the outlet to the sac.

In some skulls the continuance of the duct has almost the same diameter as the sac, and in such instances drainage could readily be had into the nasolacrimal duct; but where there is a decided constriction at the junction of the sac and duct, this junction, being encased in unyielding bone, with a plexus of blood vessels adjacent to the bone, forms an excellent point for irritation, subsequent inflammation and deranged function.

Hence, from all that has been said regarding atypical development, such as multiple punctae and canaliculae, unusual and varying junction points between the sac and the duct, multiple nasolacrimal ducts with their anomalous diverticulae and valves, and vagaries in anatomic development of nasolacrimal ostia, wherein there is no standard type of ostium, it is evident that all of these factors may enter into the causation of chronicity of infections of the tear sac and its appendages.

We have all had remarkable success in the treatment of certain cases of dacryocystitis by simple measures. Schaeffer says: "The success or failure of nonsurgical treatment of the diseased nasolacrimal passageways is largely dependent upon the anatomic type of nasolacrimal duct and ostium encountered." With this thought in view, we should study our cases and decide on "what to do and how to do it." Then use the surgical or nonsurgical procedure best adapted to the case in hand.

As to the surgical treatment, it would seem but logical to conclude that an operation which would have for its aim the

shortcircuiting of the nasolacrimal duct, by a direct opening through the sac into the nose, would be a procedure that would at once solve practically all those cases with anatomic variances and resultant pathology distal to and within the sac itself. Such an operation I wish again to bring to your attention, with some innovation in technic, which I believe warrants me in presuming upon your time and attention for this, my third paper, given before this Academy.

THE OPERATION.

A preliminary injection of morphin and atropin is given,

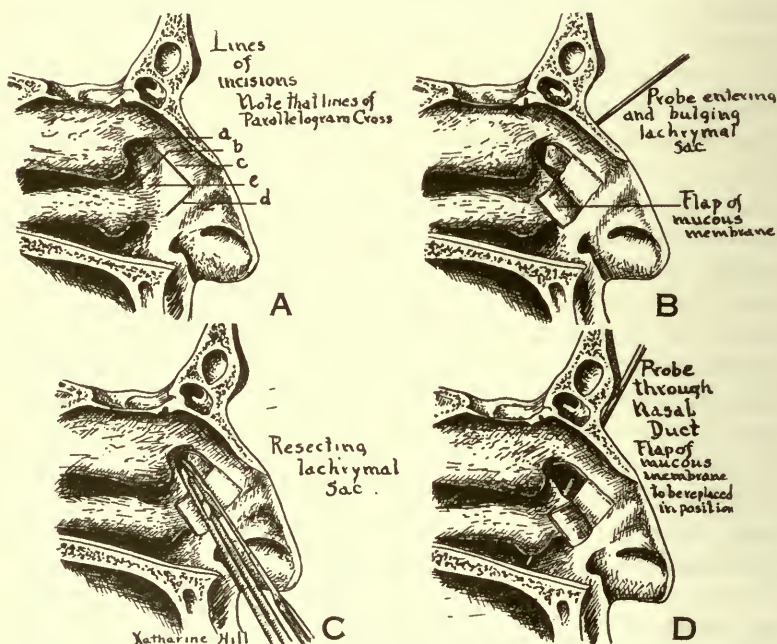


Fig. 1. Various steps in the intranasal work. Fig. C: Knife and tenaculum being used. Punch forceps has been also used to punch out sac or additional bone.

preferably one hour before the time set for the operation. The nose should be washed with normal salt solution with the patient seated, head bent forward on the chest and the mouth open. This permits a very satisfactory cleansing of the nasal chambers. The usual cleansing measures for the skin are made use of. Local anesthesia within the nose is obtained as for a submucous operation, but with greater attention paid to the upper and outer aspect of the nasal chamber. Externally, several injections of 1% novocain solution are carried down to the periosteum at points above, below and forward to the sac, as well as the skin overlying this

region. A few drops of 2% butyn should be used in the eye. To get the best anesthesia from the novocain, one should wait 5 to 20 minutes, and during this time, the incisions in the mucous membrane of the nose can be made and should be done as follows:

A parallelogram of mucoperiosteum is outlined by four incisions, which include within their borders the torus lacrimalis. Two of these incisions run vertical to the plane of the nasal bone, and the other two, the one above and the other below the torus lacrimalis, are parallel to each other, and run in the direction of the nasal bone. The upper incision is



Fig. 2. Making the 3 m.m. stab incision directly into sac with a cataract knife.

carried forward to the edge of the pyriform process, at which point another incision line is made downward parallel to the second and third lines spoken of, and this in turn enables one to turn down a flap of mucoperiosteum similar to a leaf in a book. This has the advantage of denuding the bone proximal to the point for attack with the chisel, and allows one more readily to throw the light to the point wanted, which is the torus lacrimalis, under which lies the sac.

One should make these incision lines definitely and not scratch about. It has been alleged that the intranasal methods of sac drainage are mussy, blind and comparatively inefficient. They can be made highly efficient and there is no need

for mussy work. For this reason, I have given this outline for the incision in detail. One now removes the mucoperiosteum from off the *torus lacrimalis*, and within the square which was outlined by the incisions.

At this juncture, a 3 mm. stab incision is made, with a suitable knife, directly into the sac, and a number 3 pointed Bowman probe is introduced. The sac is outlined by the probe to determine its topography and the general condition to be found. The point of the probe is then brought anteriorly along the floor of the sac to the anterior lacrimal crest.

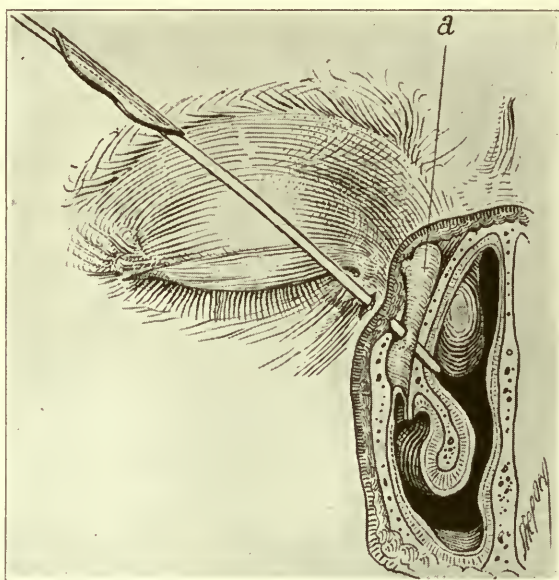


Fig. 3. Probe entering nasal cavity orientates one as to the location for the intranasal work as described.

at about the lower border of the upper third of the sac, when the probe is carried backward, pressure being made the while. When the probe comes to the thin bone, immediate entrance is made into the nasal cavity. The end of the probe, as seen within the nose, tells one where to place his chisel in order to remove the nasal process of the superior maxillary bone. The chisel is directed toward the equator of the eye on the side operated. The surgeon should control both chisel and mallet. This bone is quite dense and resists the chisel to quite a degree, so much so, that I have found it of advantage to make two preliminary nicks in the bone above and below, which outline the area to be removed and at the same time

lessen the resistance the bone offers to the chisel. Dorsalward, the bone readily fractures through, with possibly a portion of the lacrimal bone coming away with it. On examining this bone removed, one finds the smooth facet made by the sac resting thereon.

The probe is now partly withdrawn and the sac is pushed through the newly formed window opening by engaging the central portion of the sac. The sac bulges readily into the nasal chamber and may be slit anteriorly. The nasal punch may be inserted, and one rapidly punches out the lateral sac



Fig. 4. Shows the Bowman probe having punctured the thin lacrimal bone at its junction with the maxillary bone.

wall together with any additional bone desired. Again, one may use the special grasping forceps (right and left), and with the knife resect the portion of the sac overlying the opening made.

At this point, repeated washings are made through the stab wound with a lacrimal syringe filled with boracic acid solution, until the solution comes from the nostril free from blood. A small rubber tube (I use a slit infant catheter) is then inserted into the sac through the stab incision and carried downward to the anterior naris. Several syringefuls of boracic acid solution are again used. A small bit of gauze tape is carried up in the nose to assist in holding in place the flap of mucoperiosteum, which is placed in position, and the operation is finished.

One will encounter cases where it is almost impossible to see the bony point where the sac lies. This may be due to a deflected septum, a curved pyriform process, or an overhanging middle turbinal. These obstacles may be overcome

by proper preliminary surgical work. Again, in such cases I have made use of a punch chisel, which is inserted through the stab wound, and thus remove the bone from without, thereby reversing the method of attack upon the bone.

The burr has also been used by me, and this was first sug-



Fig. 5. Operation completed. Rubber tube in position and anchored by silk thread secured to forehead. Solution being washed through tube and seen escaping from nostril.

gested to me by Dr. Joseph C. Beck in his discussion of my paper, read before this Academy at its Boston Meeting in 1914. Dr. Beck made use of a round headed burr through a rather wide opening directly into the sac, whose edges were retracted by skin sutures at either side of the incision. Soon after that, I had the instrument makers, Messrs. V. Mueller & Company, make me a canula burr to be used in this connection, and of various sizes. The canula is to be introduced over an obturator apparatus protruding into the sac from the nose, to avoid displacement of the burr and consequent in-

jury to the sac walls. I will say, that I prefer the combined operation as given here, in preference to either the punch or the burr used from without.

THE AFTER CARE.

Attention to petty details in the after care is as important as the operation itself. For 72 hours the tube is maintained in position, and the irrigations are made daily by inserting the tip of the lacrimal syringe into the tube as it protrudes from the stab incision, and washing through several syringefuls of boracic acid solution. At the end of this time, the tube is withdrawn nasalward, as this tends to keep the sac edges in apposition to the nasal mucosa, thus more readily assuring agglutination of these raw surfaces, a permanent opening being the result. After the withdrawal of the tube, continued washings through the stab incision are kept up for a total period of ten days. No suture is necessary to close the incision, for its edges readily coapt. There is no resultant scar. One can maintain a small needle like opening by inserting the number 20 tip of a lacrimal syringe, having an olive tip, each morning, and thereby wash through with the boracic acid solution in the daily toilet that is made.

One great advantage of this operative procedure is that the stay in the hospital is greatly lessened. One day is sufficient, but of course, a stay of about three days is preferred, when the case can be made an ambulatory one, and the patient can then come for daily treatment for a period of a week or ten days.

CONCLUSIONS.

We are led to adopt this line of treatment mainly for two reasons and these are: That we know that in a very large percentage of the cases we will have a cure of the dacryocystitis. Furthermore, that there will be a functioning tear apparatus when we are through.

The small stab incision, being only three or four millimeters in length, permits the insertion of the sound, likewise the obturator if it is desired, and the wound may be enlarged slightly to admit the burr. As said before, I do not recommend the burr except in cases where the intranasal structures are such as to prevent one doing the intranasal work. There is no need of a stitch for closure. It also permits one more readily to explore the cavity of the sac as well as the duct, and makes it possible to insert the rubber tube readily. There is no resultant scar or cicatrization.

The after care is of great importance. The irrigations should be kept up as outlined for a period of ten days. The tube is withdrawn in three days. Attention should be paid to the opening within the nose, and granulations kept down as after a radical mastoid operation, but for a greatly lessened period of time.

The operation is indicated in epiphora, chronic dacryocystitis and fistula, and can be done in the presence of phlegmon.

DISCUSSION.

DR. JOSEPH C. BECK, Chicago: I wish to limit my remarks to the end results of these various operations, and might preface my remarks by saying that, in view of the fact that I have this disease and have not yet been operated, in my judgment the perfect operation has not yet been developed. Having operated upon quite a number of these cases and observed them carefully, I am of the opinion that the operation has not yet been developed that would suit me for operation upon my own tear duct.

The end results depend upon something the speaker did not mention, and that is the pathology of the sac. Those who support removal of the sac have good grounds to say that the sac ought to be removed, because it is not functioning, and there is a mass of granulations within the cavity, although I think it is slightly exaggerated if you say that all ducts are in that condition, because it is proven that many of these cases operated by either the intranasal or the extranasal method are successful. A good many of them retain the mucous membrane.

As to the modification of technic, everyone has his own modification. I have recently adopted a trick that Dr. Mosher uses, although not in the same operation. I retain the catheter by passing a thread from the canaliculus and fixing it by a stitch on the forehead, leaving it there for three days. In that way you have a channel that does not close up.

DR. J. SHELDON CLARK, Freeport, Ill., (closing): This paper was intended more to present matters of technic than pathologic conditions.

I hope Doctor Beck will soon decide on some operative procedure, because as it is now, I believe he has quite a sufficient number from which to choose, and surely some of them must be good.

Doctor Beck raises the question of the advisability of following this procedure in cases where there is considerable pathology, such as large masses of granulation tissue in and about the sac. I feel that in such instances the infection has occurred by reason of some anatomic defect, which in turn interferes with drainage of the sac. In all such cases, a wide opening directly from the sac into the nose, thereby short-circuiting the duct, would result in the rapid disappearance of pathology.

In the matter of diagnosis, some work has been done in this line, especially by Campbell, Carter and Doub, who make use of the Roentgen ray, following the injection of the nasolacrimal passageways with bismuth, for determining the diverticulae and other irregularities of which I speak, as well as the patency of the sac and duct.

OPHTHALMOSCOPIC FINDINGS OF VARIOUS SPECIES OF VERTEBRATES.

JOHN H. RINDLAUB, M.D.

FARGO, NORTH DAKOTA.

As a preface, at the outset, permit me to say that I have been interested in this subject a number of years, so that when I go on my annual summer vacation to the lakes, I would just as soon think of leaving my ophthalmoscope at home as I would my gun or my rod and reel. Dr. Casey Wood's enthusiasm has been contagious, and to him I owe much for creating in me an incentive to study the various fundi of animals. His work on the Fundi of Birds is a classic, and although he is the author of several systems, the American Encyclopedia of Ophthalmology, and has written extensively and well, I predict that his Fundi of Birds will remain an authority long after many of his other writings have been forgotten. For my material, I have not only drawn freely from his work, but also from such well known authorities as Lindsay Johnson, Head, Haab, Kalt, Collins, Wiekorkiewicz, Beer and others, and I wish here to make full acknowledgement of my indebtedness.

The subject selected for demonstration is a wide one. Involving as it does, patient and nonrewarding research, without thought of financial return, it has enlisted earnest and distinguished workers, and has accumulated a literature of its own which an ordinary lifetime is hardly long enough to master. It is not without a certain sense of absurdity in the undertaking, that an attempt is made to crowd such a subject into so small a space; but it will, of course, be understood that nothing more is intended than a superficial and hasty glance at its general outlines.

Some one has said that "Science is Classified Knowledge." In the study of nature much is learned by comparison. Several centuries before Christ, the Greek philosopher, Aristotle, had studied 500 species of vertebrates and many hundreds of different invertebrates, and divided them into two groups: those having blood (vertebrates) and those without blood (invertebrates)—colorless blood at that time not being recognized as such. These two groups he classified into eight subgroups.

Aristotle was descended from a long line of physicians, lived near the sea, with its infinite variety of life, with leisure and wealth, the possessor of what was then a fine library, and above all possessed of a deep philosophic and scientific spirit, which combined to produce results which were not surpassed for many centuries, and which won for him the title of Father of Natural History. It was he who discovered that all Caucasian babies have blue eyes when they are born.

Since Aristotle's time, scientists have chosen many methods of classification, some of which have proven impractical. Plainly, we could not group animals according to habitat or color, or we would have some birds, some dogs, some monkeys and many of the lower animals in the same group. When one recalls that there are millions of different animals, he can see that the task of grouping them is an enormous one. Those who have not made zoology and biology a study are apt to think of a geographic classification. One thinks of a lion as being found in Asia, the kiwi and kangaroo in Australia, the iguana in South America, and the turkey, the prairie dog, black bear, rattlesnake and the coyote in our own country, but none of these are found in Europe. The grizzly bear is found only in the Rocky Mountains, and the alligator in Florida and the swamps of the Gulf of Mexico. So you see it would be comparatively easy to construct a map showing the areas of the country in which many animals are found.

Looking at such a map, it is of keen interest to know why certain animals are confined to certain boundaries; what effect has the passing from one region to another, and how is this migration accomplished. We know that the ocean is no obstacle to many animals, yet the English sparrow, the rat or the horse would not be in America to-day were it not through the agency of man. In fact, a study of the part played by man in the distribution of other animals is a most fascinating one from its economic as well as scientific view point.

Parker and Haswell have divided the earth into faunal areas: The Arctic, the North Temperate, the South American, the Indo-African, the Madagascar, the Patagonian, and the Australian. The difference in the faunae in the different regions is due to the land and water barriers, to dispersion and to climatic conditions.

The latest authorities divide the Animal Kingdom into twelve great branches; but for the sake of simplicity, Herrick

has grouped four of these into one—the worms. There is one resemblance that all in one group bear to the others. All fishes, reptiles, birds and mammals possess what is called a notochord, or popularly known as a spinal cord, enclosed in a spinal column, or something to take the place of a backbone or vertebra, so you see eleven of the branches, and by far the greater number, have no backbone and are called invertebrates.

Although man belongs to the most highly developed branch of animals, it by no means represents the most numerous. There are over one million different species of insects, three hundred thousand of which have been studied and classified, and other species of animals are myriad in number upon the earth and in the sea.

The brain has reached its most perfect stage in the mammalian, especially in the primates, the order to which man belongs, but the same cannot be said of the human eye. As Franz points out in his text, *Comparative (microscopic) Anatomy of Vertebrates*, 1913, it is not the human eye, but the bird's eye, that stands highest in the scale, and between the so-called lowest and highest organism, one finds animal eyes that seem to be out of all proportion to the development of the other parts of the organism. However, the exceptions are all the more noticeable, because they are so few as to be almost negligible. The human eye is not only inferior in many respects to even the poorest forms of the avian eye, but probably ranks as low as the eye of the amphibians and fish. The bird's retina, if we are to judge by the number of light percipient elements to the square millimeter and by the sharp division of the layers of the retina, not to mention the exceedingly elaborate accommodative apparatus, is a much more highly developed and effective visual organ than that possessed by any race of man.

A comparison of the various organs of the visual apparatus in the class of vertebrates shows that the bird's eye is probably derived from the reptilian, or, rather, that birds and reptiles had a common ancestor. The descendants evolved a much more effective eye in the bird. The ancestor or ancestors have been grouped by Zoologists under the common name of *Sauropsida*.

Just as John Ray, in the seventeenth century, put the science of Zoology on an organized basis, by a comparison of the anatomic structures, so men like Wood and Slonaker

of America, Franz and Beer of Germany, Beauregard of France, and Lindsay Johnson, Collins and Nettleship of England, in our own time, have done much to aid in this work of systematizing the knowledge of animals, by the study of the fundus of the eye, and their researches have thrown light upon the classification of doubtful species. Indeed, their accounts of their researches furnish the most fascinating reading. If you have ever tried to study the eyes of animals in the living, you will ask yourself what methods did these men use who have examined hundreds. Lindsay Johnson, of England, whose report in the Philosophical Transactions to the Royal Society of London covers a detailed record of over 800, was fortunate in having as a friend the artist Head, who made sketches in color of the back of the eye or the fundus. At the outset it was his practice to use a muzzle or an anesthetic, but later he learned how to make use of a net, kindness, patience and coaxing, and perhaps all four, and he was enabled to examine the eyes of, as I said before, over eight hundred animals, making full notes and sketches of the fundi. Of course, no one without an enthusiastic and scientific mind and the means could ever accomplish such a monumental work. There was only a very limited edition of his work, which contained some fifty hand colored sketches, and it is said to have cost him \$30,000 to produce it.

As Casey Wood says, the great variety of beautiful pictures obtained by views of animal backgrounds with the mirror, and their value in classification, can be appreciated only by those who have examined a number of varieties of animals with the ophthalmoscope. In *wild varieties*, the colored and often brilliant picture of the background practically never varies, and it is just as possible for the expert to determine the family and sometimes the order of a Reptile, Bird, Fish or Mammal by examining its fundus oculi as it is for the artist to distinguish at a glance a Rembrandt from a Raphael, a Ruebens from a Franz Hals, or a Corot from a Turner. In domestic animals, Man, for example, the variations in the fundus appearances of individuals are marked, and the same is largely true of our house animals, whose habits and eyes undergo a variety of changes corresponding with their changed and changing environment. In the wild species, the appearances as determined by the ophthalmoscope during life are so constant, that the background picture furnishes cer-

tain data for a classification of quite as much value as the variations in any other organ.

Let us hope, that as the invention and perfection of the microtome and the improvements in microscopic methods produced a profound influence on the progress of Zoology in the last quarter of the century, so will the ophthalmoscope prove a valuable aid to the naturalist in our own time. The problems of Zoology must be approached from all sides, and if the new "Natural History" is going to be superior and more scientific, we must utilize to the full all that can be learned both in and out of the laboratory.

A series of fifty colored lantern slides of the fundi of vertebrates will be shown, which are intended to throw additional light on the subject, and from which can be drawn the following:

CONCLUSIONS.

In the study of the fundus, as in Nature, much is learned by comparison.

The red end of the spectrum is most common in the coloring of the fundus, the other colors getting rarer as you approach the violet end.

The optic disc, in a majority of vertebrates, is circular.

Among the Mammals, only the Primates possess a macula or a corresponding area.

The fundus has a distinct taxonomic value in classification.

Views of the eyegrounds of the wild varieties practically never vary.

Domestication produces changes in the fundus and other parts.

Researches have thrown light upon the systematizing of doubtful species.

Evidence obtained furnishes argument favorable to the theory of evolution.

DISCUSSION.

DR. GEORGE F. KEIPER, Lafayette, Indiana: We all ought to know something about comparative ophthalmology. To illustrate: A number of years ago I was called by Crouch & Son to inspect the eyes of one of their valuable horses, which was apparently blind. This firm deals in Belgian and Percheron horses for breeding purposes. I declined to go, because I knew nothing about the eyes of horses, and advised the firm to call a veterinary surgeon. The reply was that they would trust the eyes of their horses in no other hands except those of a competent oculist. Thus complimented, I went and inspected the eyes in question.

Then I inspected the eyes of a number of horses known to possess normal vision. This work was a revelation to me, as their eyes differ markedly from the human eye. We made a diagnosis and saved the eyesight of the particular horse in question.

Having examined the eyes of these horses, my curiosity was aroused as to the structure of such eyes and the eyes of other animals, so I mounted a number of eyes of different animals, some of which I brought with me, and are now in the exhibit room. I invite you to inspect these mounts, that you may see what the inside of these eyes look like.

Further, I would recommend the study of the comparative anatomy of the eye, that we may be helpful to the lower animals, so far as eyesight is concerned. Animals are becoming more and more valuable, as time goes on, and we ought to be able to do something to conserve and recover eyesight for them, besides being in the very desirable position of being able to teach veterinarians how to do likewise, for very few of the latter know very much of comparative ophthalmology and its practical application.

SOME REMARKS ON NYSTAGMUS.

G. W. MACKENZIE, M.D.

PHILADELPHIA, PA.

When Dr. Skillern, the Spokesman for the Program Committee, invited the writer to present a paper before the Academy, it was suggested that the subject be one, the nature of which should appeal to both Eye and Ear Specialists. A further suggestion of the Committee was to the effect that a highly technical paper, delving into speculative and fanciful theories, would be less acceptable to the average Fellow in attendance, than one which adheres more closely to fundamental and established facts. These suggestions were agreeable to the writer. Accordingly, a paper on nystagmus was prepared, in which the attempt is made to present the subject in the simplest manner possible, based mostly upon personal observations of a large number of normal individuals, and of others with definitely known pathologic conditions, covering a period of eighteen years.

We can best describe nystagmus as involuntary to and fro movements of the eyes. According to the comparative rapidity of the two opposite movements (to and fro), we may conveniently divide nystagmus into two general forms, the undulatory and the rhythmic. In the undulatory, sometimes called oscillatory, form of nystagmus, the to and fro movements occur with equal rapidity, resembling therefore the pendulum movements of a clock. Undulatory nystagmus is most commonly found in individuals suffering from impairment of vision, more especially central vision. The earlier in life the impairment of vision occurs, the more prone the individual is to develop nystagmus. The lesion responsible for the visual defect may have occurred during intrauterine life. The location of the lesion may be in the macula, the maculopapillar bundle, or the ultimate center for central vision.

It is claimed that central opacities of the optical media alone may produce nystagmus. This claim has not been entirely confirmed in the experience of the writer. In the vast majority of cases of corneal opacity, we find no evidence of nystagmus. Theoretically, the location of the opacity ought to exert some determining influence. For instance, we

should be able to find more cases of undulatory nystagmus among those individuals with central than in those with ex-central opacities. However, this is not borne out by experience, for we find in the vast majority of cases of central scars of the cornea, even the small dense ones, no sign of nystagmus. The same holds true of anterior and posterior polar cataracts. The size of the opacity appears to exert little influence, for we find quite as many cases of nystagmus among individuals with large as among those with small scars. In spite of this, there remain two apparently inconsistent facts. The first is that undulatory nystagmus is found more commonly among those individuals affected with corneal opacities, together with poor vision, than in those free from these defects. The second is that only a relatively small percent of the total number of individuals so affected manifest nystagmus. How can these two facts be harmonized and at the same time prove that opacity of the optical media is an actual cause of nystagmus? The answer is that it cannot be done. We must look elsewhere for the cause. The opacity of the optical media is a mere incident and not the cause of the nystagmus. Let us consider the following facts and draw our conclusions accordingly:

(a) The most classical cases of undulatory nystagmus are to be found in those conditions where there is a relative absence, through degeneration or otherwise, of the retinal pigment in the macular region; for instance, in albinos, coal miners, and cases of central choroiditis.

(b) In cases of diseases of the anterior part of the eyeball, the posterior part is more often diseased than in those cases where the anterior part of the eyeball has remained healthy.

(c) In cases of marked undulatory nystagmus, it is decidedly more difficult to make a detailed examination of the eyegrounds than in an eyeball that remains stationary.

(d) In those eyes with opacities of the optical media, it is more difficult to see the finer details of the fundus than in eyes presenting normally transparent media.

There is but one logical conclusion, and that is the uveal tract is more often involved in cases of opacity of the cornea than is demonstrable by ophthalmoscopic examination, and undulatory nystagmus is due more to the poor central vision from lack of function of the macula than from the impairment of vision from corneal opacity alone. In other words, the

opacity of the cornea is a mere incident and not the sole cause of the nystagmus. It is possible, too, in individuals blinded by any disease of the eye anterior to the retina, that the lessened amount of illumination reaching the retina causes a relative loss of pigmentation of the retinal epithelium.

Undulatory nystagmus is induced by the futile efforts of the patient to find that sharpest vision, which is possible in normal individuals only when the image of an external object falls upon the macula, which is impossible in the case of one whose macula is for any reason more or less functionless. The failure to accomplish the impossible, in spite of repeated efforts to do so over a prolonged period, must necessarily result in the development of habitual movements of the eyes in the form of undulatory nystagmus.

Summarizing: Undulatory nystagmus is involuntary to and fro movements of the eyes. These to and fro movements occur with *equal* rapidity. It results from visual defects, especially central. The lesion responsible for the visual defect occurs in early life or even during intrauterine life.

Rhythmic nystagmus, like the undulatory form, is involuntary to and fro movements of the eyes, but unlike the undulatory, the to and fro movements occur with *unequal* rapidity; in other words, the movement in one direction is comparatively slow, while the return movement is rapid.

In recent years, rhythmic nystagmus has been the subject of considerable study, because of its common occurrence in diseases of the so-called vestibular apparatus and its nervous pathways.

Although the writer has described both forms of nystagmus in a broad way as involuntary to and fro movements of the eyes, he wishes to qualify somewhat his earlier statement in so far as undulatory nystagmus is concerned. Every ophthalmologist can recall the difficulty he has experienced at times in studying the ocular fundi of a patient affected with undulatory nystagmus; furthermore, he can recall how at a properly directed command, the patient is able to inhibit somewhat the intensity of the ocular movements for a brief period only (several seconds), when the intensity of the movements again increases up to that which is usual for the particular patient under observation. Thus we see that undulatory nystagmus, although it is involuntary, can be more or less controlled by a strong inhibitory effort on the part of the patient. The ability of the patient to inhibit the intens-

ity of his eye movements in the case of undulatory nystagmus, is comparable with the ability of a patient to inhibit the pill rolling movements of the fingers in the case of paralysis agitans. Rhythmic nystagmus, on the contrary, cannot be inhibited by the patient, no matter how strongly he may will to do so.

The eye movements in undulatory nystagmus may occur in any plane—the frontal (tortional), sagittal (vertical), or the horizontal. Besides, these movements may occur in a combination of planes—tortional with horizontal, is perhaps the most common of all. Since, in the undulatory form of nystagmus, the to and fro movements occur with equal rapidity, we cannot consistently refer to the direction of the nystagmus, for instance, to the right or to the left, as we can in the case of rhythmic nystagmus.

Rhythmic nystagmus, no matter from what cause, remains true to form under all circumstances. This is not true of the undulatory form, for we find that it is changed to the rhythmic form when the patient attempts in the slightest degree to look in any other direction than straight ahead; especially is this the case if he looks laterally to either side. For instance, if a patient with undulatory nystagmus, the result of a central visual defect, attempts to look to the right, he will manifest a rhythmic nystagmus to the right, which just as readily changes to a rhythmic nystagmus to the left, when he attempts to look to the left. The question naturally arises, why is this so? It is so, for the reason that most, if not all, normal individuals manifest a rhythmic nystagmus to the right, when they look to the extreme right, and a rhythmic nystagmus to the left, when they look to the extreme left. This rhythmic nystagmus, which is evident in normal individuals, may be horizontal, or mixed horizontal and rotary, or rotary, depending upon how far laterally he directs his vision. It is purely physiologic so long as the intensity of the nystagmus is equal to the two sides for the same degree of angle deviation of the eyes, and disappears when the patient looks straight ahead at infinity.

This physiologic nystagmus exerts a modifying influence in every case of spontaneous nystagmus, undulatory or rhythmic; for instance, we have observed above that the influence of the physiologic element is sufficient to change the character of nystagmus from the undulatory to the rhythmic form, and this, too, by only a moderate effort at side glancing.

Again, in the case of spontaneous rhythmic nystagmus, let us say to the right, the slightest attempt to move the eyes to the right is sufficient to *increase* perceptibly the intensity of the nystagmus to the right, while the slightest attempt to move the eyes to the left will *decrease* the intensity of the nystagmus to the right. The nystagmus is increased to the right in the case just cited, because there are two influences cooperating to produce nystagmus to the right: the original one that produced the spontaneous nystagmus to the right, plus the physiologic nystagmus, or the tendency to the physiologic nystagmus, to the right when the patient looks to the right. Rhythmic nystagmus to the right is decreased when the patient looks to the left, because there are two influences operating against one another: the original one that produced the spontaneous nystagmus to the right, and the inhibiting influence of the physiologic nystagmus to the left. In some cases, the physiologic influence toward the left, when looking to the left, is sufficient to neutralize the pathologically induced spontaneous nystagmus to the right, thereby arresting it. This would happen in a case where the pathologically induced spontaneous nystagmus to the right is less pronounced than in the preceding case. In still other cases, the influence of the physiologic element toward the left may be sufficient to overbalance the pathologic element toward the right, the result of which is a rhythmic nystagmus to the left, so long as the patient looks intently in that direction. In this case, the pathologically produced spontaneous nystagmus to the right is minimal in amount but of clinical significance.

Rhythmic nystagmus, like undulatory nystagmus, may occur in any plane or combination of planes, but unlike the undulatory nystagmus, it occurs in a definite direction, and is generally designated according to the direction of its quicker component. Thus we may speak consistently of horizontal nystagmus to the left, rotary to the right, etc. To designate it less fully would be insufficient for accuracy. The moment we designate its direction, we at the same time stamp the nystagmus as rhythmic in form, for the simple reason that it is not possible to conceive of any definite direction for undulatory nystagmus, where the two movements occur with equal rapidity. With some beginners, there seems to be confusion as to how we should designate the direction of rhythmic nystagmus, due no doubt to the fact as established by Bárány, that the slow movements of the eyes is the actual vestibular

reflex, while the quicker movement is purely voluntary; for instance, if the right labyrinth and nerve are stimulated with the negative pole of the galvanic current, there follows directly a slow movement of the eyes to the left side. So long as the individual being tested is conscious, he experiences a visual sensation of external objects moving slowly to the right. To reorientate himself, he quickly brings his eyes to the right by a voluntary effort originating from an impulse in the cerebral cortex. The continued application of the negative current stimulates the right vestibular nerve; then Deiter's nucleus sends the impulse along the posterior longitudinal bundle to the eye muscle nuclei, causing the eyes again to move slowly to the left; when the brain recognizes the outside world sliding to the right, by an effort of the will the subject under examination brings his eyes back to their primary position. In short, the slow movement is the actual vestibular reflex and is, therefore, involuntary; while the quick movement is purely voluntary. In designating the direction of rhythmic nystagmus, it is customary to designate it after the direction of the quicker movement, and not according to the direction of the actual reflex movement, as some might prefer.

Rhythmic nystagmus can be produced experimentally in several different ways: (1) by directing the normal individual to look to the extreme right or extreme left, referred to earlier and termed physiologic. (2) By placing before the eye a prism base in or out of sufficient strength to throw a strain on one or the other of the horizontally acting eye muscles, when the nystagmus will occur in the plane corresponding to the action of the muscles upon which the strain is placed; the *direction* of the nystagmus (quicker component) will correspond to the normal action of the same *muscle* (i. e., toward the apex of the prism). In only a few experiments of this kind are we able to produce a typical rhythmic nystagmus, but in a sufficient number of cases to satisfy the principle. (3) By stimulating the vestibular apparatus and nerve with negative galvanism or by inhibiting the normal tonus with positive galvanism. (4) By producing endolymph impact or movement against the hair cells of the crista ampullaris of the semicircular canals, either by the so-called caloric or turning tests.

Furthermore, rhythmic nystagmus may be produced experimentally in a pathologic case; for instance, in a patient afflicted with undulatory nystagmus of ocular origin, the

undulatory character of the nystagmus may be changed to the rhythmic form by having the patient look in any other direction than straight ahead, which has already been alluded to:

Rhythmic nystagmus, pure and simple, may result from certain well known pathologic conditions. Rhythmic nystagmus may occur from paresis of any of the extraocular muscles, when it may be accompanied with vertigo. The nystagmus will take place in the same plane and the same direction as the normal action of the weak muscle. The nystagmus reaches its maximum intensity with the maximum effort to use the paretic muscle. Rhythmic nystagmus is never present in such a case when the patient looks in the opposite direction, that is, away from the paretic muscle, and is rarely, if ever, evident when the patient looks straight ahead, which is contrary to that which occurs in the next class of cases.

Rhythmic nystagmus results from pathologic conditions affecting the inner ear or eighth nerve. The character of these conditions is not always the same. They may be conveniently divided into two classes, the irritative and destructive. Again, conditions of a similar kind may differ in degree. For instance, we find irritative conditions in the inner ear, which, so far as their effects are concerned, compare with that of stimulation with the cathode. Both tend to produce a rhythmic nystagmus toward the side of irritation or stimulation. The intensity of the resulting nystagmus depends upon the intensity of the pathologic irritation or cathodal stimulation. In both instances, the resulting nystagmus is not purely horizontal, for the vertical canals are stimulated as well as the horizontal, nor is the nystagmus purely rotary, for the horizontal canal is included with the verticals in the stimulation. The resulting nystagmus from stimulation of all these canals ought to occur in both the horizontal and frontal planes, and in fact it does. In other words, there is a mixed horizontal and rotary nystagmus. If the pathologic irritation is limited to a single canal, we ought to get a single plane nystagmus, and in fact we do, observable in cases of irritative lesions (congestion) of the external semicircular canal, in the early stages of labyrinthine fistula.

In a diffuse destructive lesion of the inner ear, so long as it is of recent origin, there results a rhythmic nystagmus, mixed horizontal and rotary, away from the affected side, comparable with that produced by positive galvanism. In long

standing cases (a year or more following the destruction), the spontaneous rhythmic nystagmus that existed early will have disappeared entirely, or nearly so. In the circumscribed destructive lesions of recent origin, there occurs a spontaneous rhythmic nystagmus away from the side of the lesion, in the plane corresponding to the plane of the canal affected.

Lesions of the vestibular branch of the eighth nerve produce mixed horizontal and rotary nystagmus toward the side of the affection in cases of mild congestion, and away from the side of the lesion in those cases of inflammation sufficiently pronounced as to cause diminution or loss of function. The time limit will not permit me to indulge in this interesting subject further at this time.

Recently there has been some doubt cast upon the possibility of nystagmus resulting from lesions of the cerebellum. Notwithstanding, I am convinced that nystagmus can occur from a lesion of the cerebellum pure and simple. My reasons are many, but I will content myself with the citation of a single case at this time. In consultation with Dr. Sears, of Huntingdon, Pa., several years ago, I operated a case of right temporosphenoidal lobe abscess, complicated with a right sided labyrinthine suppuration. Dr. Sears, after a most searching examination, diagnosed correctly the labyrinthine suppuration, and felt sure as to the presence of an abscess somewhere in the brain, but was inclined to believe the abscess was in the cerebellum.

As a result of the right sided labyrinthine suppuration, the patient manifested before the operation a spontaneous mixed rotary and horizontal nystagmus to the opposite (left) side. At the operation, the labyrinth was exenterated and nothing at all was noticed about the behaviour of the eyes, the patient being under light narcosis. The cerebellum was then incised in a search for the abscess, when immediately the patient sprung a horizontal nystagmus to the right (operated) side of exceptionally wide excursions. A few minutes later the temporosphenoidal lobe was incised and a large quantity of foul smelling pus was emptied from the abscess cavity, which produced no further effect upon the nystagmus. In brief, the right sided labyrinthine suppuration had caused, as it always does, a rhythmic mixed horizontal and rotary nystagmus toward the opposite (left) side; exenteration of the labyrinth produced no change in the existing nystagmus. Incision of the right cerebellar hemisphere immediately changed the

plane and direction of the nystagmus from a mixed horizontal and rotary toward the left to a purely horizontal toward the right. Further operative work in the middle fossa did not influence the character of the nystagmus produced shortly before, by incision of the cerebellum. The irritation produced in the cerebellum by the incision was the clearest evidence to me and those present, that a lesion of the cerebellum alone will produce nystagmus; others have observed it, too.

Speaking of nystagmus of cerebellar origin, the writer has observed it to be more frequently purely horizontal, less frequently purely vertical, and least frequently mixed horizontal and rotary. Purely horizontal nystagmus, when of cerebellar origin, points toward a lesion of one of the hemispheres. Since abscess of the cerebellum occurs more frequently in one of the hemispheres than in the middle lobe or vermis, we naturally find horizontal nystagmus to be the prevailing form. In the earliest stages, that is, the period of congestion, when we have signs of irritation, the stimulating effects of the irritation produces horizontal nystagmus toward the side of the lesion: very soon the effects of destruction are felt, when the nystagmus swings over toward the opposite side (away from the side of the lesion); however, so long as the process is limited to one hemisphere, the nystagmus retains its horizontal character. In diseases of the vermis alone, for instance a small circumscribed tumor, the nystagmus is purely vertical. Since abscess of the cerebellum is rarely if ever limited to the vermis, a vertical nystagmus associated with increased intracranial pressure would point toward a tumor rather than an abscess. Rarely does one find a mixed rotary and horizontal nystagmus in abscess or tumor of the cerebellum; when it does occur, it is not due to the lesion in the cerebellum proper, but to pressure on the root of the vestibular branch of the eighth nerve or its nuclei; in other words, it is a more or less distant working symptom in the sense that MacEwen first intended its interpretation.

For the benefit of the eye men, the writer feels that he would like to call attention to the fact that nystagmus can be readily observed during the making of an ophthalmoscopic examination, especially by the direct method. Every now and then, one may observe rhythmic nystagmus movements of the eye at the posterior pole, where its direction is the reverse of what it is at the anterior pole. In all such cases, a note

should be made of the fact, and the case studied more closely with the object of determining its cause.

With one exception, I have deliberately avoided the citation of cases, as interesting as they are. My intention has been to carry out as far as possible the wishes of the Program Committee, who feel as the writer does, that the greatest need is to the greatest number—that it is better to present a more simple paper, at the risk of making it kindergarten in style, rather than attempt the more technical one, that might appeal to but a limited few.

DISCUSSION.

PROF. ROBERT BÁRÁNY, Upsala University, Sweden: Mr. Chairman and Gentlemen: First I wish to thank the gentleman for his most friendly words bestowed upon me. I also wish to thank you for the invitation you were so kind as to send me at Upsala. I am glad to come to America and to see this country, and I wish only to say that I am very grateful for your reception.

Now as to the paper of Doctor Mackenzie: He has mentioned something in regard to the registration of nystagmus. There has been quite a lot of work done in registering the nystagmus of patients, especially by Ohm, and some interesting things have come out of that. But we find even more interesting the registration of nystagmus, particularly from the muscles, after the method which Bartels has indicated. Bartels was the first to try to register nystagmus from the muscles after the enucleation of the eyeball, but he has done this only upon the rectus externus and internus. He has there found very remarkable things—nystagmus to the right and to the left from one muscle only. He has found that if the animal be narcotised, nystagmus in one muscle often disappears and only one muscle shows nystagmus. It is very remarkable that both these movements can be done if only one muscle is acting and the other is not severed.

Doctor Mackenzie has said that the quick component of nystagmus is of cortical origin. I do not know whether he attributed that to me or not, but I never said it. That is an error. It has been known since Hgyes' work in 1881, that one can take away the whole cortex cerebri in rabbits and the nystagmus will still be present. These experiments have been done by Kreidl and Karper on monkeys, and by Magnus and other men in Utrecht on rabbits, dogs, cats, and monkeys. These investigations have absolutely proven, that in nystagmus neither movement is associated with the cortex. Both movements are localized in the brain stem. Wilson and Pike of Chicago have also published a paper on that, and have come to the conclusion that the rapid movement is from the cortex, but that is not correct. They have overlooked shock in the animal. If the animal is in a state of shock, this rapid movement will not be seen, but if one does the operation so that no shock occurs, one can see the quick component.

The examination of the vertical and rotatory nystagmus by the registration of the isolated muscles is very interesting. I have done

that in a number of rabbits and found very curious things. The rectus superior, rectus inferior, obliquus superior, and obliquus inferior muscles are very often not working together as one should expect theoretically—often only one is working and the others are quiet. Often one obliquus is contracting, and one rectus is relaxing. Often the amount of contraction or relaxation is very different in the antagonists. My investigations are not complete—it would be necessary to study these things quite in detail. But it should be possible, also, to study these things in the human being, if one does an enucleation, and therefore I am speaking of these things before a joint meeting. I, myself, made some investigations in Copenhagen. We did enucleations in two cases under local anesthesia, and it was easy to investigate the nystagmus by syringing. I think quite a lot of new things could be found, if one would pay attention in this operation to the action of the muscles. If you cannot irritate the labyrinth, you can produce a nystagmus which I call "railroad" nystagmus—the nystagmus one has on the railway when one looks out of the window. This nystagmus can be produced by turning a cylinder with black and white stripes upon it before the eyes of the patient. Without the least inconvenience, you can try the patient if you let him look at the cylinder with his sound eye, and you could in this way investigate the muscles of the enucleated eye.

I also wish to say a few words about the inhibition of the nystagmus. There, also, I cannot quite agree with Doctor Mackenzie. He said undulatory nystagmus can be inhibited by fixation, but not vestibular nystagmus. That is not correct. Horizontal rhythmic nystagmus can be inhibited by fixation. This was shown by Brener in 1874. I have therefore always advocated, in the examination of the turning nystagmus, the use of opaque spectacles before the eyes of the patient, because otherwise you get an inhibition of the nystagmus. A very simple way to get an inhibition of the vestibular nystagmus has been described by one of my assistants, Dr. Nasiell, in Upsala. I had first found it, but he examined quite a number of cases and found it absolutely typical. Every sort of rhythmic or undulatory nystagmus can be inhibited strongly by closure of the eyelids. The moment you open the eyes, the nystagmus is present as before. The explanation is, that if you close the eyes strongly, you innervate all the muscles of the eye simultaneously. You also get contraction of the pupil at the same time, and the nystagmus is mechanically inhibited.

In an article in the Laryngoscope the last year, I have stated that rotatory rhythmic nystagmus can be inhibited by strong convergence. I can do it myself, and I have examined other people who have strong convergence, who could also do it. The explanation is simple. You know, in adduction of the eye, the rotatory nystagmus is made by the rectus superior and inferior and not by the obliqui. The obliqui are elevators and depressors in this position of the eye, and the rectus superior and inferior are at the same time adductors, and therefore if you very strongly innervate the convergence, the rectus inferior and superior will be simultaneously innervated, together with the rectus internus, and you therefore get an inhibition of the rotatory nystagmus.

Now the question of nystagmus from cerebellar lesion: You know that Neumann and I in 1906 described this nystagmus from cerebellar abscess. It was the same observation which Doctor Mackenzie now has reported—not quite identical, but very nearly so. It was a case which first had destruction of the labyrinth on the right, and consequently nystagmus to the left side. The labyrinth was operated, but the nystagmus was not changed. One day afterward, the patient developed a very strong nystagmus to the operated side, and when I saw that I said, "This nystagmus cannot come from the labyrinth, it must be of intracranial origin." The patient was operated and had a cerebellar abscess. This was the first case. Afterwards, Neumann and I found that this is a very typical and regular observation in cerebellar abscesses. The question is whether the nystagmus is really developed from the cerebellum, or by the pressure of the abscess in the cerebellum upon the medulla oblongata. This question has not been solved. It will not be cleared through the case which Doctor Mackenzie has reported, because if you do an incision of the cerebellum, it is clear that the pressure is changed in the posterior fossa, and because of this change of pressure, nystagmus suddenly arises. So I do not believe that it is proven that the incision directly caused the nystagmus. In rabbits, of course, you can produce nystagmus from the nuclei of the cerebellum. We are quite sure of that from experiments. But how it is in man has not yet been established. Leidler, who has done very good and important work on nystagmus from the cerebellum and medulla oblongata, believes the nystagmus is not due to the cerebellum, but that it is possible that from the nuclei of the cerebellum there might arise a nystagmus also in mankind. I am not sure of that, because the cases have not been proven sufficiently.

DR. GEORGE W. MACKENZIE (closing): I wish to thank Professor Bárány for discussing my paper. I will speak, so far as the time permits, on the objections he raised.

To begin with, the paper was not intended to be one delving into the deepest problems of nystagmus. The effort was rather to present the subject in a manner calculated to interest a larger number of the fellows than is possible with the more technical paper. In other words, the object was to popularize the subject. A great deal has been written on the subject of nystagmus, some of which is true, but a great deal more that is merely speculative. This has resulted in a good deal of confusion, and the beginner, when he glances at the literature, is prone to say "What's the use?" Otologists have gotten into bad repute with the neurologist on this very subject, because of too much guesswork. The paper is an appeal to those Fellows of the Academy who have done little or no work of the kind in the past.

The first question raised by Professor Bárány is that of not caring to have assigned to him the responsibility for the idea that the quick component of nystagmus is due to a cortical influence. He further states that with experiments upon animals, the reaction of the quick and slow component is the same whether the cerebrum is intact or not. In answer to this, if we take a patient narcotized with ether, or one unconscious from uremia, or one stupid from brain abscess or any other

condition capable of producing partial or total unconsciousness, he will manifest merely a conjugate deviation of the eyes. If the deviation is to the right, in the comatose condition, when you waken him he will manifest a rhythmic nystagmus to the opposite (left) side. This clinical observation has been made repeatedly, and by many observers. In view of this fact, I want to ask Professor Bárány whether the quick component was voluntary or not? Next I want to ask if we know any part of the brain where there are voluntary centers other than the cerebral cortex? In experiments upon animals, we cannot invariably come to the same conclusions as in the human, because we find in the evolution of the human being quite a number of differences occurring. We are not compelled to accept the physiologic experiments on animals as conclusive for humans, especially when it comes to the consideration of the higher functions.

I will not be allowed enough time to answer all the questions Professor Bárány has raised, and therefore am at a disadvantage, but I would like to speak of some other points he raised.

He claims that in a patient with hemiplegia, with a double sided lesion (a condition which must be exceedingly rare), the presence of a nystagmus is evidence that a cortical impulse could have nothing to do with the quick movement. Let us take an individual who is perfectly normal, and make a turning test for horizontal nystagmus; after ten or fifteen turns to the right, there follows a definite horizontal nystagmus to the left side. There occurs a slow movement to the right side and a quick movement to the left side. When timing the after nystagmus, it is important to note the movements up to the point when they cease; otherwise a mistake will be made and the time will be too short. Near the termination of the test, a very slow movement of the eyes to the right may be seen by the keen observer. When we give the patient a command to look straight ahead, a quick movement occurs, which is the last or nearly the last one. I want to know if anything else than a voluntary effort on the part of the patient could have produced the quick movement in a perfectly conscious normal individual. Further, we at the present time know of no other location for voluntary centers than in the cerebral cortex.

I recall the case that Neumann and Bárány worked on, where they found nystagmus to the affected side after it had been away from the affected side. This case of Doctor Sears is not exactly the same type of case as Neumann's. The case that Professors Neumann and Bárány were working on was a clinical case in which symptoms of destruction of the labyrinth were present, causing a rhythmic nystagmus away from the side of the labyrinth destruction; then suddenly the patient showed a nystagmus towards the side of the lesion. The Sears' case was different in this particular, that removal of the labyrinth alone made no change in the mixed rotary and horizontal nystagmus toward the unaffected side, proving that it had existed prior to and independently of the operation. Immediately following the incision into the cerebellum, there was a wide excursion horizontal nystagmus to the same (affected) side. What else could have brought about this striking change in the direction and plane of the nystagmus than the incision of the cerebellum? We later opened a large abscess of the right temporosphenoidal lobe,

but it produced no further change in the nystagmus. So I leave it to you, gentlemen, whether the incision of the cerebellum produced it or not.

Professor Bárány disputes my claim concerning the inability of one to inhibit rhythmic nystagmus, and as proof of his contention points to the observation that nystagmus is inhibited by convergence of the eyes, and by forcible closure of the eyelids. It may be that we misunderstand one another, for further on in the paper I said that in the case of spontaneous rhythmic nystagmus to the right, the slightest attempt to move the eyes to the right is sufficient to *increase* the nystagmus to the right, while the slightest attempt to move the eyes to the left will *decrease* the intensity of the nystagmus to the right. The decrease of the intensity of the nystagmus to the right is not an inhibition in the sense that I referred to above. It is rather a positive force working in a contrary direction, which exerts a neutralizing influence on the nystagmus, and not an inhibiting one induced by a negative force.

The claim that forced closure of the eyelids inhibits nystagmus I will answer in the same way. If the nystagmus is decreased by forced closure of the lids, it is accomplished not by inhibition, which is a negative influence, but by force, which is a positive one. I do not wish to deny the possibility of decreasing nystagmus by forced closure of the eyelid, but for the life of me I fail to see how it is possible by finger palpation to make accurate observations of delicate eye movements through the puckered up, thickened tissues of the lids.

PROF. ROBERT BÁRÁNY: I think it better to answer the question immediately. It is surely a very puzzling and important question. It is quite sure, that in man also the quick component of the nystagmus does not belong to the cortex. You know, that if one is in coma, he may also have no pupillary reflexes and no corneal reflexes. Nobody thinks the pupillary reflex is due to the cortex. We are quite sure, that at the same time the cortex is paralyzed, the deeper centers in the medulla might in many cases also be affected, and this one must think is the case in a deep narcosis. If you have a narcosis which is deep enough, the centers in the medulla oblongata are paralyzed. If you go very far with the narcosis, you get death by paralyzing the centers in the medulla oblongata. So it is quite sure that these centers can be paralyzed by the narcosis, and in case the patient is in coma, they can also be out of function. I could prove that in man also the quick component is not due to the cortex, in a case who had hemiplegia and paralysis of the associated movements of the eyes to one side. If a man has hemiplegia due to a lesion of the right cerebral hemisphere, he cannot look to the left, and the eyes, if he tries to look to the left, go only to the midline. If you now produce a nystagmus to the left, you will get a perfectly normal quick component, but this component goes only to the midline, because the voluntary action which should carry it to the other side is lacking. But it is an absolutely normal quick component in this case. I have seen a case who had hemiplegia on both sides and who could not look to either side. It was not complete paralysis, but strong paresis of the look to both sides. This patient had a perfectly normal quick component to both sides. So I believe that these reasonings are sufficient evidence that the quick component is not due to the cortex.

THE END RESULTS OF RADICAL OPERATIONS ON THE ACCESSORY SINUSES.

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I do not know how often the question has been put to me, "Skillern, now as man to man, what is the ultimate condition of the patient after a radical on the frontal, ethmoid?" or whatever the sinus under discussion may be. As the frequency of this interrogation bespeaks its importance, I purpose, with your indulgence, to base this dissertation upon the answer, at least as far as my own personal experience is concerned.

First of all, what is a radical operation on a sinus? I take it, that when one speaks of a radical operation, one means an operation performed with the single thought in mind of an absolute relief from the symptoms with a more or less perfect cure, regardless of the severity or extent of the surgical procedure. The last sentence expresses that which not infrequently takes place, in that the operation is oftentimes performed regardless of its extent or severity. That this expression has been repeated, thus overly placing emphasis upon it, is intentional and deservedly so, as did these not obtain, the question as to the ultimate results would never have been trite but theoretic, and only of academic interest instead of representing the oft too trying reality, which not infrequently arises in after months to plague those of us who are called upon to operate upon these cavities accessory to the nasal passages. When one considers the degrees in the severity of infection as well as the pathologic changes which have occurred, it is an extremely difficult task to approach this subject without entering into the details and finer minutiae, so that for the sake of clarity let us waive aside all so-called errors of judgment as to the advisability of the operation, overenthusiasm of the operator, or last resort methods, and assume that a radical operation was clearly indicated, but despite its timely application, the end results were not everything to be desired. What do we mean when we say the end results were not everything to be desired? As these differ markedly with the different sinuses, it will be best to consider them from their separate and distinct standpoints.

FRONTAL SINUS.

It is presupposed in this sinus, that an external operation through the brow with removal of one or more of the walls has been performed. What untoward sequelae of a more or less permanent nature may follow?

1. Persistence of the pain.
2. Hemianesthesia of the brow and scalp.
3. Persistence of the discharge.
4. Neuralgia about the cicatrix.
5. Diplopia.
6. Epiphora.

I am purposely omitting the severe forms such as meningitis, blindness, fistula formation, etc., as our discourse is limited to the minor conditions, which however are of sufficient annoyance to the patient to prevent the operation from being termed a complete success. Patients are only too prone to forget the previous severity of their disease and dwell upon some present condition, which would have been considered trivial had they suddenly been transformed from one to the other, but after all it is only human nature, and there is not one of us here that would not feel similarly disposed, were the personalities reversed and he were the patient instead of the physician. When a patient undergoes an operation, he expects to be cured and all cured, and should some slight trace of the old trouble linger or a new symptom appear, it is bound to be a source of dissatisfaction and a very real dissatisfaction at that, but if we could only make them realize that the surgical intervention was the means of saving or prolonging their lives, or at least rescuing them from a state of chronic invalidism, and they could also be made to understand that the chances of complete recovery are always inversely and not directly to the extent and severity of the operation, the surgeon's task would be far easier than as at present constituted. To revert then to the untoward sequelae, let us for a moment consider the first one, that of *persistence of the pain*. After an external operation on the frontal sinus, if the pain persists, to be perfectly frank, either the operation was incompletely carried out and a focus of infection permitted to remain, or the pain is due to some other sinus or indeed to some other condition which may be quite remote. I recall distinctly two patients whom my operation failed to cure of their headaches. Although both had chronic sinusitis, one

subsequently was permanently relieved after his acidosis had been eliminated, while with the other, the gynecologist succeeded where I had failed. So in these cases where pain is the principal complaint, while the external operation will in the vast majority of instances bring about complete relief, nevertheless the prognosis should not be entirely Utopian in its promises.

Hemianesthesia of the brow and scalp. This condition always manifests itself immediately following the operation, due to the severance of the supraorbital nerve close to its exit from the foramen, and is always mentioned by the patient, more it is true in the order of passing than as an actual complaint. Fortunately, the anesthesia is of temporary duration, as in the course of a few weeks regeneration of the nerves or a collateral anastomosis occurs, with a return of full sensibility to the previously affected area. I have never seen a case where this has become permanent.

Persistence of the discharge. If I were asked to name the most frequent untoward sequela of an external radical on the frontal sinus, I would unhesitatingly choose this one. In the natural course of events, a purulent discharge always follows, which gradually assumes more and more a mucoid character, at the same time becoming less and less, until in the course of weeks it ceases altogether. Unfortunately, in a great many cases this discharge continues much longer than we care to see it, despite appropriate treatments. If in the quiet of our study we review our operation, small wonder will it appear that such a secretion makes its appearance, for it represents Nature's attempt to throw off those portions of tissue which have been devitalized by our curettes and cutting forceps. When we consider the bony cell walls, periosteum and adjacent mucosa which has been crunched, scraped and lacerated, together with the ravages of the disease, much regeneration of tissue must occur before these parts again assume their earlier functioning activities. The persistence of this discharge will be directly as the extent of the surgical application, and should this have embraced large and remote areas, just that much longer will it require before complete regeneration occurs, if indeed it ever does. In any event, to use a classic phrase, a locus minoris resistantiae is established, which may manifest itself with every exposure to cold and wet, or indeed any cause which would be conducive to an acute coryza. However, in the advent of such an eventuality, all criticism must

be instantly swept aside, when comparison is made with the previous condition of the patient. Who of us would not gladly exchange the dangers and distress of a chronic suppurating frontal sinus with inadequate drainage for an intermittent mucoid discharge with no subjective symptoms, which moreover is gradually becoming less and less with the lapse of time? I am confident we would be unanimous on this point.

Neuralgia about the cicatrix. In my experience this is a relatively rare occurrence, and when manifesting itself is due to the severed nerve ends becoming involved in the newly formed cicatricial tissue. Under the influence of massage, it usually disappears in the course of time, and I have never seen a case that required a subsequent resection for liberation of the enmeshed nerve filaments.

Diplopia. Despite the assurance of Hajek, double vision is very apt to occur when the tendon of the superior oblique is elevated and its osseous support removed, unless special measures are taken to prevent it. In many of my earlier cases, permanent diplopia developed, especially on looking downward, as for example descending a stairway, which required lenses to correct the defective vision. For the past several years, after completion of the operation, I have caught up the loosened tendon at its point of contact with the pulley, and throwing a fine catgut ligature around it, tied it to the bridge of bone, approximately in its original position. Since practicing this procedure, diplopia has manifested itself in but one case, and curiously the muscle on the operated side appeared the stronger when examined by the ophthalmologist. This was due to my placing too much tension on the tendon while bringing it into position, and then tying it too tight. We have now profited by this experience, and while making it snug, are careful not to put it under any tension. At this point, I would like to report a unique experience which happened in the early summer, a few months ago, with the first case on which I performed a Killian operation, in 1908. This girl developed a marked diplopia, which obliged her to constantly wear glasses. Fourteen years later, the sinus again became infected. The condition refusing to respond to conservative methods, it necessitated a reopening of the affected area. After curetting away a small portion of diseased bone, the tendon of the muscle was caught and tied as in the present method. I was particularly interested to learn what effect

this would have on the longstanding diplopia. You can imagine my feeling of satisfaction when, on removing the bandages, she said "Why I no longer see double," and subsequent examination showed that she had fused binocular vision, which has remained so ever since.

Epiphora. When one considers that the lacrimal sac is elevated from its bed and the underlying bone resected, it is to be marveled that tearing does not invariably follow the radical frontal operation. As a matter of fact, it usually does if one takes the trouble to make a test, but fortunately it seems to be of but temporary duration and disappears shortly after the patient leaves the hospital. I have referred patients to the ophthalmologist immediately following the operation, who reported that it was difficult to force a passage for fluids through the membranous canal. On reexamination, in three or four weeks, syringing succeeded with no difficulty whatever, showing that nature had restored the physiologic function of the canal as a drainage passage for the tears into the nose.

Now to return to our subject of end results, how many patients are troubled with these unpleasant sequelae, and how great is their degree of discomfort? I am not prepared to give definite statistics, but certainly less than 10 percent have any of these one year after the operation. The most frequent is the tendency toward catching cold, with a discharge, but even this in time becomes less noticeable, and for all I know ceases entirely. We do not perform the radical operation (except in a few instances such as threatened cerebral or orbital complications) until all conservative means have failed; but when indicated, it is one of the most satisfactory methods of relieving and ultimately curing a severe case of chronic frontal sinusitis that we have at our command.

MAXILLARY SINUS.

The radical operation on this sinus, when properly performed, is seldom followed by unpleasant after effects. Certain false steps however may be the cause of one of the following:

1. Anesthesia of the upper lip and teeth on the operated side.
2. Permanent fistula formation into the mouth.
3. Excessive dryness of the nose on the affected side.
4. Gradual return of the discharge after an apparent cure.

Anesthesia of the lip and upper teeth. The loss of sensibility of the lip due to cutting the inferior branch of the infraorbital nerve frequently occurs, but is only of temporary duration, as full sensibility soon returns. Anesthesia of the teeth is a more serious matter and should never occur, as it is due to faulty technic. It will be remembered that the supradental nerve runs just above and parallel to the roots of the teeth, and it is never necessary to resect the bone to this extent, unless it is absolutely indicated; then the teeth are no longer viable and should be extracted. Permanent anesthesia of the teeth means that the nerve has been injured or destroyed, and usually presages the ultimate loss of the teeth, except under very favorable circumstances and in the hands of a first class dentist.

Permanent fistula formation into the mouth. This unfortunate sequela usually results from making the incision too far down on the gums, thus not having sufficient redundant tissue on closure of the wound. There is no necessity for this and it can always be avoided, provided of course that a previous fistula did not exist. As the permanent closure of these fistulas is almost a chapter in itself, time is insufficient to enter into that phase of the discussion.

Excessive dryness of the nose and affected side. A rare eventuality, unless one is prodigal in removing the soft tissue of the inferior turbinate. I have rarely found it necessary to remove much of this structure, and now our routine procedure is to remove only that portion of the lower edge anteriorly that obstructs free aeration of the sinus, after the counter-opening is made into the nose.

Gradual return of the discharge after an apparent cure. This disappointing result is usually due to insufficient removal of the diseased lining membrane of the sinus, in which permanent pathologic changes have occurred. In my experience, this omission takes place in the prelacrimar recess, which can only be reached by a right angled curette. It is surprising, while using this instrument, to note the large amount of polypoid tissue that can be contained in such a comparatively small area. Another cause of failure is making too small a counter-opening into the nose. I believe for the best results, this opening should extend from the anterior end of the turbinate to about its middle, and from its insertion well into the floor of the nose. It must be remembered that not only drainage but aeration is required, and I question very much whether the latter is not

just as important as the former in healing these old antral suppurations.

SPHENOID SINUS.

The important sequelae which are connected with this cavity are:

1. Gradual closure of the opening before the suppuration has ceased.
2. Reinfection, with intermittent suppuration.

Gradual narrowing with incomplete closure of the opening. This is observed in practically every case following operation on the sphenoid, and is undoubtedly an effort of Nature to restore the parts to their original condition. In most cases, this is an advantage rather than a disadvantage, if we presuppose that the sinus possesses some physiologic function.

Reinfection with intermittent suppuration is not an infrequent occurrence and usually succumbs readily after a slight enlargement of the ostium, which at this time is but a minor office procedure.

ETHMOID LABYRINTH.

The postoperative behavior of these cells depends upon so many factors, that this phase of sinuology represents to-day perhaps the most important in the entire category of paranasal affections. New methods of operating have sprung up here and there and been hailed as the last word, only to die away and fall into discard, or become so modified as to entirely lose their original identity. This fact alone shows that of all the methods hitherto devised, none have proven a *vade mecum* for all classes of cases. How many of us have seen cases of rather mild and decidedly bearable ethmoiditis made almost unbearable and well nigh hopeless by injudicious or incomplete operations! I know of no sinus condition where it is easier to advise operation and more difficult to extricate ourselves after this badly advised piece of surgery has been consummated than this very condition. How often has an anatomically perfect, though mildly infected, labyrinth been transformed into a disorganized suppurating mass, which defies the best efforts of even a master to change it back to its former morphologic configuration, with its normal physiologic activities! A great many more times, even in our own practices, I dare say, than we would care to admit. Let us then consider more in detail the end results often following partial

and complete ethmoidectomy, with certain reasons pertaining thereto.

1. Continuance of the discharge.
2. Continuance of the pain.
3. Partial occlusion of the nostril.
4. Ocular symptoms which did not obtain previous to the operation.

Continuance of the discharge. Of all the postoperative conditions one is called upon to treat, this one I believe comprises more than all the rest combined. Accepting this statement at its face value, what reason can be attributed for the prevalence of the discharge following operations? In answering this question, we can, as the Chinaman says, talk "goody, goody" or tell the truth. If we choose the more difficult way, we will say it was because we did not eradicate the disease at the time of operation, and furthermore broke into healthy tissue, which also became infected, so that the end result was worse than the original condition. In analyzing such a case, it was not altogether our fault, for we proceeded according to our best judgment, removing the cells as they appeared to be diseased. It is just here, however, that we made our error both of judgment and of technic, for one can not differentiate by inspection through a nostril, diseased ethmoid tissue which might regenerate from that which imperatively must be removed. I think the preliminary resection of the middle turbinate followed by appropriate treatment and careful observation before further surgical procedure is adopted will very largely solve our ethmoidal problem.

Continuation of the pain. By this I mean headaches associated with an ethmoid infection continue both in their frequency and severity. It must be remembered that many other factors may enter here, and the hypothesis that our operation was incomplete and failed to remove the cause of the headaches may be without foundation, as these pains might easily have some other genesis. In view of a more or less wide experience with this class of cases, I have ceased to take these complaints as seriously as formerly, and now look for extrinsic causes (if I am convinced the ethmoid is not at fault) usually with success.

Partial occlusion of the nostril is mentioned only in passing, and is due to carelessness in permitting synechiae to form, thus interfering in a greater or less degree with the respiratory air current passing through the nostril.

Ocular symptoms which did not prevail previous to the operation. This is fortunately a very rare occurrence, even though

only the thickness of a sheet of paper separates the orbital fat from the ethmoidal mucosa. I have frequently seen the conjunctiva become so engorged that it protruded between the lids, following extensive operative measures on the ethmoid, only to again recede and finally disappear, without leaving any untoward effects. Patients occasionally complain of a weakness of the eye, but this must be classed in the nature of a neurosis, for the ophthalmologist always returns negative reports. I do not recall ever having seen a case of serious disturbance of vision following an intranasal operation, although such cases have occasionally been reported. In reviewing our ethmoidal work, I am sure most of us will agree that it is best to make haste slowly and not try to cure the patient in one fell swoop. It is far wiser to remove the middle turbinate as a preliminary operation, not only for the purpose of permitting better aeration and the furtherance of drainage, but what is even more important, to allow comprehensive study of the labyrinth, which was an impossibility with this structure in situ. Finally, let us remember that experience has taught us that radical operations upon the accessory sinuses do not always spell radical cures.

A NEW METHOD OF RADICAL OPERATION FOR CHRONIC EAR SUPPURATION.

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It may seem presumptuous to come with a new radical operation, since a radical operation has now been done for thirty years. The bone operation is not new. It may be new only in the degree, that the procedure used in this operation is peculiar to me, and has not been used by other men, so far as I know. I will at once describe the details.

In 1911, I recommended that, in the radical operation, one should be very careful with the membranous meatus, so that it should not be injured during the operation. Since 1911, I do not remove the membranous meatus, but perform the operation around it. I take away the bone around the meatus with precisely the same care that you take away the bone from the dura, and I do not injure it in the operation. I have done this with the purpose of not destroying something which before the operation has been healthy. If one does the operation in the old way, removing the membranous meatus from the bone, or forcibly employs a retractor, or retracts the split meatus with a gauze strip, it is very likely that he will injure the epidermis in the meatus, especially the epidermis of the anterior wall, which can so easily be destroyed. If, furthermore, a single bone chip should fall into the tympanic cavity, it might destroy the epidermis of the meatus on the back of the tympanum, which I would like to preserve.

But, after completing the so-called conservative radical operation, with conservation of the drum and the ossicles, I always used to do the plastic of the meatus, as, naturally, also in the ordinary radical operation.

In November, 1921, I had to operate on a case where, before the operation, I could not determine whether the case was acute or chronic, and I said, "We will see during the operation what should be done." I operated and found disease in the attic, and removed the attic wall completely, but without injuring the membranous meatus. Then I removed the incus, but not the drum or the hammer, and at this stage of the operation it came to my mind whether it is really necessary to do a plastic, or whether it might not be possible to treat the case like a simple

mastoid. When I got this idea, I immediately followed it out and did not do a plastic. I simply put in a cigarette drain of thin India rubber filled with gauze, and sutured the skin over the bone opening up to the inferior angle. After one day, I removed the cigarette drain and treated it with guttapercha paper. Now, I found in this case no suppuration, but only a very little serous secretion, and the wound behind the ear healed very quickly. This case, however, afterward developed badly, as it was a tuberculous one, and there was a fatal outcome. This was the first case in which I did this operation, but I had quite a number of cases afterward, cholesteatoma cases and others. I have now done this operation—the radical operation without plastic on the meatus—in more than thirty cases, and can only say that it gives very good results.

As to your procedure in any given case: It is quite clear that you must pay attention to the special conditions of the case. The bone operation can be the same, but the treatment of the tympanic cavity, the drum, and the ossicles varies. If I have a perforation of the attic and a cholesteatoma, with an intact drum, the incus in connection with the stapes, and the hearing very good, then I always do a so-called conservative radical operation. I do a complete radical operation, but do not remove the ossicles and the drum; only the cholesteatoma is thoroughly syringed and curetted out. When the operation is completed, I have a normal meatus, with ossicles and drum intact and only a little hollow in the attic.

And now what happens? The whole cavity fills with a blood clot alongside of the cigarette drain, and on the second day, as one removes the latter, the cavity fills completely with blood. If there is no secretion, no further drainage behind the ear is necessary, and the case may be healed completely in six days. The little perforation in the attic granulates from the edges and epidermis grows over it, and in six to ten days it is closed and there is a normal meatus and normal drum, and no one can suspect that there ever had been a chronic suppuration. This is the most favorable case. One of my cases healed practically in this way, the only difference from this ideal case was, that the patient before the operation had a labyrinthitis which destroyed the hearing, and that I removed the incus, and that the head of the hammer also was destroyed. But the case was healed in ten days with closure of the attic perforation.*

*I have recently operated on a case of Dr. Pfingsten, of St. Louis, with the same ideal result. The case had an attic perforation, but no cholesteatoma.

Another of my cases had a perforation in the posterior part of the drum up to the antrum. The hammer was intact and the cholesteatoma was behind the hammer. This case also was completely healed in two weeks, and the perforation also closed completely with epidermis.

You know that in the ordinary radical operation you have very often very hard work to keep out the granulations. Some grow from the attic, some from the facial ridge, and you have quite often great trouble to overcome the tendency of the granulations to reunite. In this operation this is just what we wish to happen, for as soon as the granulations at this narrow space have reunited, the mastoid cavity is completely divided from the tympanic cavity, and in case of a recurrence in the tympanic cavity, it is quite impossible for it to break through into the mastoid. In these cases, the cure is complete and there cannot be any further mastoid complications. This is the most favorable thing which can occur.

In other cases of attic perforation, the latter does not close, but gets much wider after the operation, because there are only soft tissues and the edges of the perforation are drawn back by the cicatrix, so that the perforation extends. I have seen cases in which the hole has become six or eight times as large as before the operation. In these cases I have seen that the division of the tympanic cavity and mastoid occurred, and that the epidermis grew out and closed the cavity completely from the mastoid. I must say that, with few exceptions, the cholesteatoma cases and chronic cases in general are healed with less discharge than the acute cases, because the infection in the chronic cases is not so virulent as in the acute. If you do this drainage, you find many cases with no suppuration but only a serous exudate, and within six days the healing takes place behind the ear in a great many instances, while the tympanic cavity requires two weeks. I have had ten cases which required seventeen or nineteen days before healing was complete—I do not remember the exact number. Naturally, in the chronic ear diseases, it is clear that we cannot expect all cases to heal in quite the same way. If you have destruction of the drum and mucous lining of the tympanic cavity, you will not have healing so quickly in the tympanic cavity. In these cases, if the mucous lining grows into the mastoid cavity, the blood clot in the mastoid cavity breaks down partially and a larger cavity in the mastoid may result. Mucous lining will grow in partly in the mastoid, but it does not matter if the mastoid is filled in its apex and deeper parts. You have

then the condition which you often see in dry perforations of the membrane, where the mucous lining is in the tympanic cavity and reaches into the antrum.

It would be too much to speak of all the possibilities that can happen in these cases, but in all the cases I have operated since last November, I have used this method and have had excellent results from it.

I want to mention a little thing before I finish. You may ask what will you do if the whole tympanic cavity is diseased, if you have to scrape out granulations from the hypotympanum, and if you have to do a labyrinth operation. In one case I had to take away granulations from the hypotympanum. This I did from the meatus and partly from behind, depressing the membranous meatus. In this way, an inspection of the tympanic cavity is often possible, and granulations can be thus removed. The diseased bone in the tympanic cavity I did not remove. If the case is not tuberculous, this will heal spontaneously after more or less time. In tuberculous cases, I recommend fresh air and general sun bath treatments after the operation.

In one case I had to do a labyrinth operation, and here I did a temporary plastic. First I removed the facial ridge as deep as possible. Then I made a cut along the facial ridge and another vertical cut behind the auricle, and then I could turn the flap upwards and retain it with a hook. Then I was able to inspect the tympanic cavity very well, and did the labyrinth operation without injuring the membranous meatus. When I finished, I laid the flap back, and it united with the edges at the facial ridge, and was a normal meatus after a few days.

So I see no cases in which I cannot use this method, and I find the healing much more rapid than in the old radical operation. This method also has the following advantage over the old operation. After the old radical, you have a large cavity and cholesteatoma reforms there often after the operation, so that the patient always must go to the physician for treatment—he cannot treat his own ear. If you employ this operation, the patient can treat his ear simply by syringing and drying, as if no operation had been performed.

DISCUSSION.

DR. H. I. LILLIE, Rochester, Minn.: It has been a great privilege indeed to listen to the paper of the great master. While listening, it occurred to me that there were several points of particular interest. Ordinarily, the justification for any operation of this kind is to control the otitis media and to preserve the hearing function. The more conservative the operative procedure to accomplish this result, the better directed is the management. The natural tendency toward the radical operation is always very conservative, quite similar to what Professor Bárány has described. We must also remember that all cases of chronic suppurative otitis media do not require operative interference. The cases must be individualized and well classified and studied before any operation is instituted. We would classify his group in those that do not require the classical radical operation, but the so-called conservative radical.

One point I did not quite understand. He stated that the cicatrix closed over the antrum by this operation, or this process, and separated the mastoid cavity from the middle ear. If the middle ear remains patent, with the Eustachian tube open, with normal function of the Eustachian tube except for the perforation, it occurred to me that it would likely keep the antrum open, as we see in cases operated for acute mastoiditis.

Another question is, what would keep these cavities from becoming reinfected? In the radical operation, the antrum and middle ear being converted into one cavity, the reinfection takes place through the tube and is easy to manage. I should like to have Professor Bárány discuss this point more in detail.

We have known of the so-called conservative radical operations, but for the most part, I believe, the philosophy and indications have not been generally accepted. Does the Professor wish to imply that all chronic conditions may be handled in this manner, and that the results are uniformly good?

DR. S. H. LARGE, Cleveland, Ohio: I wish to congratulate Professor Bárány on this new operation. I think it certainly is a great improvement on the old radical. I think most of us, in our own radical cases, when we do the operation, must tell our patients that this is not the end. Practically all of my patients return from time to time to have the meatus cleaned out. I think this is due chiefly to the injury of the membrane of the meatus. It has been found out, by examination of the membrane that has formed after the plastic, that this new membrane is not true epithelium. If you have destroyed the true epithelium of the meatus, you never get the same epithelium that you had before the operation. It is mostly scar tissue, and I really feel that Professor Bárány has opened up a wonderful field for us in this modified radical operation.

DR. JOSEPH C. BECK, Chicago, Illinois: I am so sorry that I did not get this instruction from the pastmaster, Professor Stacke, who taught me the radical operation. Naturally, I followed all these years the method I was taught in performing this operation, and like all the men have had trouble in treating the cases. The operation is much more

simple as he describes it than the Heath, or the Stacke, or the various modifications, and to hear from our guest the universally satisfactory results he has obtained makes us all feel badly because we did not know this, and have not practiced it since 1911. We had the opportunity to try it, and I stand here guilty of not trying it, although I am accused of trying everything. (Laughter). I have tried similar things in operating on so-called subacute cases.

Professor Bárány spoke of a case he could not differentiate. We do not have a time limit in these cases, but judge by the gross appearance at the time of operation. I do use the so-called modified blood clot method, but instead of leaving it open, I use the posterior stab wound for drainage, so often used at Rochester, and when that has been done in a case it has come out very well. I knew nothing about this being new, but those cases recovered.

I want to say, that for the younger men who are coming on, it will be a wonderful thing to try.

I would like to know what is the longest period of time that has elapsed in any of these cases since operation, and also what Professor Bárány will do in intracranial conditions in which a radical mastoid operation must be performed?

DR. OTTO GLOGAU, New York, N. Y.: In our work, we were governed by the idea that epithelization was necessary in places where no epithelium was needed. We never thought of it in acute mastoid cases, where the cavity fills out with connective tissue. Professor Bárány's operation takes us back to the logic, that no epithelium is needed in the middle ear, even after a radical operation. I have tried a similar method in three cases, in a nervous woman and two children. There I did not add the plastic, but I did injure the end of the canal with the idea of having the epithelium from there grow into the middle ear cavity, adhering to the old superstition. I think his idea not to injure the canal at all, but to let the whole thing fill out with connective tissue is the most logical one. One point especially appeals to me, the closing of the drum when a perforation in Shrapnell's membrane is present. If we succeed in making a conservative radical operation and let the wound close up behind, if we then succeed in removing the decayed part from the attic and the drum heals perfectly, the ideal result is obtained. I think we should thank Professor Bárány for bringing this method before us.

DR. JOHN F. BARNHILL, Indianapolis, Indiana: I hesitate very greatly to speak my convictions of the paper just presented by our eminent visitor, and discussed by the able members who have preceded me. However, I believe that not to issue some sort of warning against an indiscriminate employment of the operation proposed by Professor Bárány, would be wrong to our younger members who are striving for the best.

I think it probable, that if the Professor had had unlimited time, he would have stated that the operation he described is applicable only to a very limited number of carefully selected cases, for it seems certain if we use it in all cases that come to us, we must surely fail in a large percentage of all. If anything in our surgical specialty has been proven in the last twenty-five years, it is that thoroughness in mastoid work, in

cholesteatoma cases especially, is necessary, and to go back to surgery that is not complete, as the Professor's operation clearly seems to do, would be a very long step backward. Surgeons of wide experience, who have temporarily left the proven, standard plan of complete mastoid surgery, where indicated, to follow the modified operation of Heath and Bondy, only to return again to sane principles of operating, are not likely again to lightly consider the operation here proposed. There are, all must admit, cases to which this operation is rightly applicable, just as the Heath operation and other modified operations are applicable, but the fact undoubtedly remains that there are many others, in which all these modified radical operations would be strongly contraindicated, and I dare say the Professor will, in his closing argument, agree with me in this contention.

DR. A. H. ANDREWS, Chicago, Illinois: I am very glad to have heard this discussion by Professor Bárány. I am going to be very careful in the future with the meatus, but I do not understand how a cholesteatoma involving the whole mastoid can be cured by this procedure. If it can be cured by such an operation, then my ideas on the subject have been entirely wrong. I would like to ask Professor Bárány if ten months, which I understand is the longest period in any of his operations, is sufficient to tell what the operation will accomplish.

DR. J. A. PRATT, Minneapolis, Minn.: It is rather embarrassing to stand here and say that you have been doing an operation similar to this, especially when it comes from such a master as Professor Bárány. For the last five years, instead of doing a radical operation, except in cases of cholesteatoma, I have been doing what I call a complete simple mastoidectomy. When I read my paper describing this operation before the North Dakota State Medical Association last June, Dr. John H. Rindlaub of Fargo, in discussing it, stated that he had been performing a similar operation in place of the radical for some time. Undoubtedly many operators have been using the same technic. It is just as the Professor suggested, if a complete mastoidectomy is performed with cleaning out of all the mastoid cells, antrum, and aditus ad antrum, with curettment of the attic if necessary, we have better results than with the radical operation. I started this technic with the electric burr, which I use in all my mastoid work. Seventy-five percent of the cases have cleaned up in my hands.

I feel highly complimented that I have been doing something similar to Professor Bárány's operation. My paper on this subject appeared in the *Journal-Lancet*, September 1, 1922.

DR. GEORGE W. MACKENZIE, Philadelphia, Pa.: The criticism I have to offer of this operation is the same as that which was brought forward at Boston in 1912 regarding the Heath operation, from which Bárány's operation differs but very slightly. It may be recalled, that at that time Mr. Heath, of England, reported that a vast majority of chronic middle ear suppurations were healed by his conservative radical operation on the mastoid, leaving the middle ear untouched. He believed that in the vast majority of cases of chronic middle ear suppuration, the infection was generally limited to the mastoid cells. Dr. Yankauer, of New York, reported quite as many cases of cures by curettage of the Eustachian tube. Dr. Evelyn Nagle, of Boston, at the same Congress, reported a

remarkable number of cures of chronic middle ear suppuration by way of vaccines alone. These three authorities differed considerably in their conclusions as to the cause of the chronic middle ear suppuration and their methods of cure. One believed that the mastoid was the important seat of infection, tending to keep the chronic middle ear suppuration chronic. Another believed that the Eustachian tube was responsible, and the third thought that it was a matter of poor resistance. Mr. Heath was asked, and I repeat the question to Professor Bárány, what are the indications and contraindications for the modified radical mastoid operation as a substitute for the so-called radical? Mr. Heath failed to give them then. Perhaps Professor Bárány will give them to-day.

I disagree with Dr. Beck in recommending a modified radical mastoid operation to the younger men, for the reason that I believe it would be safer for them, so far as the ultimate results are concerned, to stick to the more radical operation.

In closing, I would like to ask Professor Bárány the question as to why there is more probability of a recurrence of a cholesteatoma when the mastoid is operated after the ordinary method than after his method, providing the operator was equally careful in each instance. Again, I would like to ask why the results should be more favorable in a case of cholesteatoma after his method than after the ordinary radical, in those cases where the operator has removed the cholesteatoma less efficiently than in the former instance?

PROFESSOR ROBERT BÁRÁNY, Upsala University, Sweden (closing): I can perhaps summarize what I have to say. I have heard it stated, that I do not recommend this operation in all the cases. I say once again, that I have not, up to the present, seen one single case where I did not have a better result than in the old radical operation. So I must say that I hold to this and recommend this method in all cases. It might be, that with more experience, in some cases it will be found not to give a satisfactory result, so that the procedure must be modified occasionally, but to do the old radical with the plastic I believe will never be necessary.

Now, as to how I operate: Naturally, I remove the whole cholesteatoma with the greatest care. You must be absolutely sure that none of the cholesteatoma remains. That is the first condition. It is easy to do that in the mastoid, but not so easy in the antrum, attic, and tympanic cavity, and you must have the best possible light for that. It is impossible to do it with a headlight on the forehead. One must use a light with the source of illumination very near the eyes—like the Klaar headlight or the Kirstein lamp. This is very important for this procedure. If you clean out the cholesteatoma perfectly, it is quite improbable that reinfection will take place, because the cavity is filled with the blood clot and later with the connective tissue. In the old operation, you had the cavity epidermized, and in that way cholesteatoma again arises, but in this way you have no epidermis left behind, and if you have done the operation well, the whole cavity will fill as in a simple mastoid operation.

In the majority of cholesteatoma cases, the Eustachian tube is closed before the operation, and you do not have to open it. If the tube is not closed, you have a mucous lining in the tympanic cavity,

and it is a question whether the division between the mastoid cavity and tympanic cavity will take place. Naturally, it is not impossible that you will have a cavity, and with that extending into the mastoid, you can get suppuration from this cavity, but I believe it is no worse than in the ordinary radical operation, because it can be drained through the tympanic cavity. I have not seen a case where you had such a big cavity as in the ordinary radical operation. In most of the cases you can, with the probe, prove that the tympanic cavity is closed in the direction of the mastoid cavity.

As to the intracranial complications, if you have a brain abscess somewhere, it is not necessary that you do a plastic operation on the meatus. You do not want a plastic, because you have to treat the brain abscess. In such a case, you simply must take care that the meatus does not get tamponed in your dressings. I should recommend to tampon the meatus daily, or to have a rubber tube in during the time you are treating this complication, but it is not necessary to do the plastic. The plastic was done to avoid getting a stenosis, and this you do not get if you proceed in my way.

DEEP INFILTRATION ANESTHESIA IN OPHTHALMIC SURGERY.

A. S. GREEN, M.D. AND L. D. GREEN, M.D.

SAN FRANCISCO, CAL.

The production of ocular anesthesia by the instillation of cocain in the cul-de-sac has been one of the greatest contributions to ophthalmic surgery. It has enabled ophthalmologists to perform operations upon the anterior segment of the eye, in non-inflammatory conditions, with little or no pain. Since its introduction by Koller in 1884, many new anesthetics have been discovered, so that now a very satisfactory anesthesia may be obtained for operations upon the conjunctiva, cornea, sclera and extraocular muscles in noninflammatory conditions by the use of a solution of cocain, holocain, alypin, butyn and various other anesthetics, either alone or in combination. But in performing an iridectomy, even when no inflammation exists, it is often impossible to operate without producing more or less severe pain. This is of extreme importance, particularly in cataract operations.

When surgery is indicated upon an inflamed eye or for enucleation, it will often happen that it is practically impossible to obtain sufficient anesthesia by instillation. A step forward was made when instillation was augmented by subconjunctival infiltration. Thus Snellen, Haab, Ellis and others performed enucleations after local subconjunctival infiltration, but they practically all had a high percentage of painful cases. As they used the conjunctival tract for obtaining their anesthesia, they soon found this method was not very effective, especially for inflamed eyes. The reason for this will be understood, if we consider what occurs in this procedure. Cocain or its substitutes instilled in the eye, act but very slightly upon the inflamed and hyperemic conjunctiva. The needle is thus introduced through a sensitive membrane, the nerve filaments of which have been at best but imperfectly anesthetized. The subjacent globe is still more sensitive, so that the traction and pressure produces severe pain. Very little absorption of the anesthetic occurs in such an eye. Thus the patient has already suffered great pain before the actual and more painful operation is even attempted. The fault lies not with the anesthetics but with the methods employed. In non-inflammatory cases, of course, the infiltration through the con-

junctiva is much more successful, but even then not entirely devoid of pain in many cases, and not sufficiently effective for certain operations. Lowenstein, of Prague, after considerable investigation, published in 1908 a still further advance in ocular anesthesia. His method consisted of injecting cocaine with a long needle through the conjunctiva.

The method which appeals most to the writers is the deep anesthesia of the globe, described by Duverger and published in the "Presse Médicale" in 1918. Duverger employs it for enucleation, evisceration, amputation of the anterior segment, and also in operations for acute or subacute glaucoma, whether performing an iridectomy or sclerectomy. It differs from the methods of inducing deep ocular anesthesia as attempted by Lowenstein, who used *cocain*, in that Duverger, who used novocain, enters the orbit through the skin and not through the conjunctiva and uses a shorter needle. He is thus enabled to reach both motor and sensory nerves of the eyeball without disturbing the inflamed and painful conjunctiva, and without traumatizing the optic nerve. The writers found this anesthesia so effective in operations for acute and chronic glaucoma, whether for trephining or iridectomies, in secondary glaucoma from plastic iritis, enucleations, muscle operations, and for the severe pain produced by subconjunctival injection of mercurial solution, that they finally gathered sufficient courage to employ it for cataract operations. The wisdom of employing so radical a method of producing anesthesia will be questioned by many who believe that they can obtain sufficient anesthesia by local instillation. We have not been so fortunate, though at various times employing the different methods of local anesthesia recommended by operators in this country and abroad.

Vitreous is frequently lost and many eyes jeopardized, while some have been sacrificed, in performing the iridectomy for a cataract operation, by the involuntary traction of the recti muscles. Of course, the blame has generally been placed upon the "unruly patient" and not upon the insufficient anesthesia. While it is true that the majority of cataracts can be removed by the ordinary local anesthesia, a certain percentage of nervous patients, made more so by their oncoming blindness, can not be sufficiently anesthetized without jeopardizing the eye. Granting that the anesthesia is complete in a large proportion of cases, we are not justified in taking a chance with the remaining. We have found fewer unruly patients since adopting the method now employed,

and would consider it taking a retrogressive step were we forced to return to the usual procedure of anesthesia for cataract surgery.

But in addition to abolishing ocular pain during the operation, we have also sought to eliminate the squeezing action of the lids. We long ago found that lid hooks and certain specula, while effective in keeping the lids away from the globe, were not sufficient to prevent the action of the brow, or the attempted action of the lids, even if held by an assistant. This often produces a reflex traction by the extraocular muscles, that will annul the operator's best efforts. With the globe, lids and brow sufficiently anesthetized, the success of the operation depends almost entirely upon the operator. The objection will probably be raised that the method is not devoid of danger from panophthalmitis or even meningitis. If rigid asepsis and antisepsis are carried out in preparing the skin and sterilizing the syringe and needle, no trouble is apt to follow. We have not had the slightest untoward effect from the deep orbital injection, and only once had a slight swelling over the brow, following repeated insertions of the needle in attempting to localize the supraorbital foramen.

The technic which we have employed on several hundred cases in the past year, is as follows: The skin of the lids and that surrounding the orbit, covering an area about 3 inches in diameter, is thoroughly sterilized by first wiping it with benzin to remove the grease, followed by a thorough application of a 3 percent tinct. of iodine, and finally finishing with removing as much of the iodine as possible with alcohol. Great care must be taken to do this well, so that no infection will follow the introduction of the needle into the orbit. It is our custom to shave the brow the night before operating. The anesthetic we use for injection is a 2 percent novocain with adrenalin solution, put up in ampules. A needle, 4 to 4½ cm. long, attached to a Luer syringe, is introduced through the skin and pushed to its full length into the orbit along its floor, directing the needle slightly inward and upward. About 1 cc. of solution is then injected, and the remaining 1 cc. is injected as the needle is gradually withdrawn and the barrel emptied. Care must be used that the needle is not withdrawn too far before finishing the injection, as in that case one will give a subconjunctival injection instead of one exclusively intraorbital, and the effect will not be nearly

so good. The point of entrance through the skin may be found by locating the outer lower rim of the orbit with the sterilized finger, so that the needle will follow a path between the inferior and external recti muscles. This is the only region where the needle may be introduced without meeting more or less undesirable obstructions. The routes recommended by some writers are anatomically objectionable.



Illustrating point of insertion of needle and its direction slightly upward and nasally, about midway between the external and inferior recti muscles. The needle, 4 to 5 cm. in length, is pushed all the way in.

By the route here followed, it is easy to reach the branches of the ciliary ganglion with a 4 to 4½ cm. needle. The short and long ciliary nerves, and thus the entire eyeball, are anesthetized. In addition, the recti muscles also are partly inhibited in their action. This is a great advantage, as it reduces the power of their backward traction, which is a frequent cause of loss of vitreous. The palpebral fissure widens,

the eye becomes more prominent and does not tend to shift about, making the operation much easier.

The strength and amount of solution here given is the minimum required. For enucleations, eviscerations and operations for acute glaucoma, a larger quantity and a stronger solution, will be necessary. (Duverger frequently uses 2 to 3 cc. of a 4 percent solution.) The next step is to anesthetize the lids and external canthus. The needle is introduced external to and about $\frac{1}{2}$ inch from the external canthus, pushed upward and inward towards the supraorbital notch, injecting gradually as the needle is pushed along. About 2 cc. is then injected into the lower lid, retaining a small amount to be injected into the external canthus. This is done to anesthetize the facial nerve in this region, and also that an external canthotomy may be performed if the palpebral fissure is small, the lids tense or press against the globe at the external canthus when the lids are separated, as occurs in some cases with prominent eyeballs. Under such complete anesthesia, the patient will be unable to quite close the lids and they will remain open from 3 to 5 mm. The anesthesia is complete in forty-five minutes, and will remain so for at least two hours. The danger lies in operating too soon following the injection, as it takes a considerable length of time for the solution to filter through the structures within the orbit.

In conclusion, we wish to emphasize that in the deep injection of the orbit, and with the proper aseptic and antiseptic precautions, we have a safe method of anesthetizing the globe and its adnexae, so that we can largely do away with general anesthesia in ophthalmic surgery. And finally, in our hands, it has proved of greatest importance in the extraction of cataracts.

LOCAL ANESTHETICS; THE PREVENTION AND TREATMENT OF POISONING THEREFROM.

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Any paper having to do with the subject of local anesthesia in oto-laryngology can have no more appropriate introduction, than an unqualified endorsement of the splendid work that Doctors Emil Mayer, Ross Hall Skillern and Robert Sonnenschein have done in the past three years, in conjunction with the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the A. M. A. The labor involved was enormous. The results should be most helpful to every oto-laryngologist.

Just as the report of this Committee, "By happy fortuitous circumstance" was made to the Section on Laryngology of the A. M. A. in New Orleans, the home of Prof. Rudolph Matas who, in the words of Dr. Mayer, has done more to establish the value of local anesthesia than any other American surgeon, so also is Minneapolis, the home of Dr. Robert Emmett Farr, whose work in this field has achieved national distinction, a most appropriate place for the presentation of any paper on this subject.

From the above, it would seem that several facts of prime importance stand out in bold relief:

- I. THAT THERE IS NO STANDARDIZED APPROVED TECHNIC FOR LOCAL ANESTHESIA IN OTO-LARYNGOLOGY.
- II. THAT CERTAIN FATALITIES ARE INEVITABLE AND UN-AVOIDABLE.
- III. THE NEED OF:
 - (A) PROPHYLAXIS AGAINST INTOXICATION FOLLOWING LOCAL ANESTHESIA.
 - (B) TREATMENT FOR INTOXICATION DURING LOCAL ANESTHESIA.

A meeting of this size provides better opportunity, in many respects, for a thorough discussion of these questions than a questionnaire, especially if a considerable number of the members of the Academy will report whatever data they can collect from their own practice, one year hence. The experience of colleagues and the views of physiologic chemists and toxicologists can be profitably included. The epitomized conclusions, published by the Academy and broadcasted throughout the country by its members, would be of great value to them, as well as the profession at large.

I. THE LACK OF STANDARDIZED APPROVED TECHNIC FOR LOCAL ANESTHESIA IN OTO-LARYNGOLOGY.

One may find sufficient authority in the literature to use any of the following drugs, either singly or in combination with adrenalin, and with or without preliminary morphin hyperdermatic injection:

Cocain	Benzol-Alcohol	Butyn
Procain	Stovain	Quinin-Urea
Apothesin	Nirvanin	Magnesium sulphat
Beta-Eucain	Allocaïn	

The concentration varies as much as 100% in some instances, cocain, for example, being used in 1/10% to 10% solutions.

The amount of drug administered also varies widely. The writer has personally seen a full ounce of 20% cocain solution routinely used for submucous resections, cotton pledgets being wrung out of this and the nasal chambers tightly packed with these for a full half hour before the operation was begun. On the other hand, a single grain of flake dissolved in six drops of adrenalin, carefully applied, produces satisfactory ischemia and complete anesthesia for certain operators.

The method of application also differs greatly. Many paint the pillars generously and fearlessly with 10% to 20% cocain solution, preparatory to infiltration. Others infiltrate only.

Following such widely varying technic in the administration of highly toxic drugs, it is only reasonable to expect occasional fatalities. Indeed, that they do not occur more frequently, under these circumstances, is truly remarkable.

II. THAT CERTAIN FATALITIES ARE INEVITABLE AND UN-AVOIDABLE.

A concrete example will best illustrate this fact. Two or three drops of 5% cocain solution were instilled into the eye of the wife of a physician, for the removal of a foreign body that had deeply imbedded itself in her cornea, in spite of her husband's warning to the oculist that a previous susceptibility had been definitely shown, when the drug had been used in combination with homatropin for refraction. Almost immediately, quite alarming toxic symptoms appeared. They were chargeable directly to idiosyncrasy. Cocain and all other local anesthetic agents are drugs, against which a certain few individuals exhibit this strange and dangerous systemic reaction. Had a tonsillectomy, submucous resection, turbinotomy or sac extirpation instead of a refraction been done at first, a fatality would almost certainly have occurred, "Inevitably and unavoidably," as

there were no clinical data at that time upon which to base even a suspicion of the existence of this condition. Fortunately, these cases are very rare. They are in direct contrast to the patient who goes through with his local anesthetic without so much as "blinking his eye."

Local anesthesia in tonsillectomy seems especially hazardous when the fatalities here are compared with the results in general surgery, and other fields. Braun, Matas, Allen, Farr, and others have reported series of cases running well into thousands, in which amounts of the solution were used that would make even the boldest oto-laryngologist tremble, and have encountered no serious difficulty. A proctologist, with whose work the writer is very familiar, used 2% procain freely with neither fear nor trouble. Urologists have found the bladder practically immune, but poisoning frequently occurs from the urethra. Many serious intoxications have followed spinal anesthesia. Ophthalmologists almost never meet with any difficulty. We rarely see or hear of any untoward results with submucous resections. The explanation of these peculiar regional differences of toxicity probably lies in the varying vascularity of the parts, and the varying rate with which the drug is passed into the general circulation.

III. (A) PROPHYLAXIS AGAINST INTOXICATION FOLLOWING LOCAL ANESTHESIA.

The occurrence of symptoms of acute poisoning—either grave or mild—together with the realization that *death* may occur almost *immediately*, are apt to strike the operator and his assistants with panic, especially if they are not thoroughly equipped to meet the emergency promptly. A fatality under such circumstances is most deplorable. It is easy to understand how it can readily become a frank invitation for malpractice litigation. The same fact was true of chloroform deaths, which nearly always occurred on the operating table. This, no doubt, has been an important factor in the almost universal discontinuance of its use for general anesthesia by American surgeons. In marked contradistinction, ether deaths rarely occur suddenly (with the exception of status lymphaticus cases), and consequently it enjoys almost complete immunity from criticism and law suits.

All operative cases should be *hospitalized*. Preliminary hypodermic injection of *morphin* and *atropin* seems to have established its usefulness, especially in nervous individuals and children. The *recumbent position* during operation is always desirable.

Braun has conclusively shown, in which opinion Matas concurs fully, that the *retardation of circulation* in the anesthetized area *prolongs* and possibly increases the local action of the drug, and correspondingly decreases its general systemic effect, because of its much slower entrance into the general blood stream. This principle has long been known to the profession, as well as to the laity. It has found widespread, practical application in the use of the Esmarch constriction in the treatment of snake and tarantula bites. Freezing the tissues produces the same effect.

Neither of these procedures can be utilized in our work. *Adrenalin*, however, forms an excellent substitute. Its action, while not as complete as an Esmarch constricting a leg or finger, nevertheless, very quickly and effectually whitens the nasal or pharyngeal mucosa after one or two swabbings. It is well to remember here that a *very few drops* are sufficient; larger amounts are unnecessary and may be *dangerous*. In addition to this preliminary application, the anesthetic solution itself, that is to be either locally applied or hypodermatically injected, should also carry an appropriate complement of adrenalin.

Cocain and procain are today the favorite local anesthetics of the vast majority of American oto-laryngologists. Both are powerful protoplasmic stimulants primarily, and paralyzants secondarily. The former is believed to be about five to eight times the more toxic. The latter produces little or no vascular constriction, and diffuses through mucous membrane with great difficulty. Cocain, on the other hand, is readily absorbed by any mucosa, a fact which has long been made use of by the "white wings" or "flake snuffers." These unfortunates frequently feel the general systemic stimulating action of the drug within one minute. It is, consequently, the anesthetic "par excellence" for *local application*. Procain, because of its lower toxicity, satisfactory anesthesia and the tolerance with which it is borne by the tissues, is ideal for *infiltration purposes*.

Whenever the personal history discloses some suspicion of idiosyncrasy, some other plan of procedure should be devised, and the reason carefully and definitely noted in the record.

Application of cocain in the nose or throat should always be preceded by thoroughly *drying* the field with cotton applicators or suction, or both. This precaution prevents the patient from swallowing any of the drug in the saliva, and delivers the solution to the area to be anesthetized undiluted by regional secretions. In the writer's opinion, however, *preliminary pharyngeal cocain applications are dangerous and unnecessary*.

The solution should be made up freshly by the *operator himself* from carefully weighed and plainly labeled powders, or from tablets that are known to have been prepared and stamped with equal precision. They may be made isotonic by the addition of sodium chlorid for better tissue tolerance. Bicarbonat of sodium is said to increase the efficiency of cocain.

The Scotchman's inflexible rule for his own golf score and general expenses—the very least possible—should always prevail; only the smallest amount of the weakest solution of the least toxic drug that experience has shown will produce satisfactory ischemia and complete anesthesia, should be administered. Common usage today more generally approves of cocain applications and procain infiltrations than any other agent, although weak (1/10%) cocain solution are probably just as safe in the latter instance. Combining local anesthetics seems to increase their toxicity. Infiltrations should always be made slowly, with the *needle receding* to avoid intravascular injection. The operator should never become too sanguine. Neither he nor the patient should ever allow themselves to be convinced that nose and throat operations under local anesthesia are ever absolutely without DANGER. All intoxications or cases that exhibit any unusual features should be immediately reported.

III. (B) TREATMENT OF INTOXICATION.

The symptoms group themselves in three stages: excitation, convulsions and paralysis.

Recently the author asked a number of oto-laryngologists "what do you do for acute cocain intoxication?" An almost totally different answer was given by each man. The majority had encountered no trouble. None mentioned the use of ether by the drop method, which promises to become an almost specific antidote. Nevertheless, the writer believes that mild intoxications are seen very frequently. They consist at first of an unusual inclination to talk, rapid breathing, slight increase in pulse rate and volume. These symptoms are due to the stimulative effect of the drug, and rapidly give place to loud laughing, crying and loss of self control, together with muscular twitchings, rapid, thready pulse and widely dilated pupils. The paralyzant action quickly makes itself manifest in definite convulsions, irregular, shallow, rapid respirations and embarrassed circulation. This sequence is usually very rapid. Most reported fatalities have occurred within a few minutes. Following Engstadt's suggestion, the author has used ether by the open drop method with great satisfaction in one of his own tonsillectomies that exhibited mild but definite symptoms of poisoning. He knows of its successful ad-

ministration in a procain intoxication in the practice of a urologist colleague, during an operation on the urethra.

A very loose open ether mask should be used to insure an abundance of oxygen. Light second stage anesthesia should be continued for several minutes. On regaining consciousness, all signs of poisoning will have disappeared. In this connection, it is interesting to recall that Katz and Meltzer found that small doses of cocain immediately stopped the whining, restlessness and excitement shown by experimental animals coming out of ether, thus showing a perfect *reverse antidosis*.

The patient should be kept *lying down*, with the head lowered. Inasmuch as excretion is probably largely through the liver, high hot colonic flushings are indicated. Otherwise the treatment is *symptomatic*. *Stimulants* should be used for cardiac or respiratory embarrassment. Amyl nitrit, aromatic spirits of ammonia, camphorated oil and strychnia should be tried. Artificial respiration and possibly intratracheal oxygen insufflation may be resorted to if breathing stops. *Sedatives*—morphin perhaps better than any other—help to quiet when the nervous element predominates.

CONCLUSIONS:

I. Idiosyncrasy inevitably provides an ever present unavoidable element of danger in local anesthesia in oto-laryngology.

II. The patient and the operator both need a standardized technic for the induction of local anesthesia, as well as a recognized rational prophylaxis against poisoning. These should bear the stamp of approval of some national body, as a guarantee of safety and effectiveness.

DISCUSSION: ON ANESTHESIA SYMPOSIUM.

DR. H. V. WÜRDEMAN, Seattle, Wash.: Regional anesthesia, to give it the proper name, is not only appropriate but may become the choice of anesthesia for nearly all major operations upon the eye, its adnexae, and the nasal sinuses. An important feature that should not be lost sight of is the psychology of the patient. Those who are overanxious, nervous or hypersensitive may also receive hyosein-morphin analgesia—and this in preference to a general anesthetic—with the exception of young children, in whom complete abolition of sensory and voluntary reflexes is desirable.

In the early part of 1894, Dr. Casey A. Wood called my attention to a letter from Dr. Cholewa of Berlin, who was enthusiastic upon a new method of anesthesia, and had been personally operated upon by the alleged discoverer, Schleich, of Berlin, whose reports were submitted to the German Congress of Surgeons in August, 1894. Dr. Wood published Cholewa's letter in the Journal of the A. M. A., May 26th,

1894, and this was followed by several exhaustive articles by myself the same year, in which were reported the results of fifty-eight operations by other surgeons, as well as myself, and demonstrations before several medical societies.

I give this brief history, for it now transpires that the original discoverer, introducer and author, was Professor Reclus of the Paris Faculté, who introduced infiltration anesthesia in 1890. The author of "Schmerzlose Operation" appropriated, (stole, if you will), the method and published it four years later, without giving credit to the distinguished Frenchman.

There is a difference between infiltration and regional anesthesia. In the method of Reclus (endermatic) appropriated by Schleich, the anesthetic is applied to the terminals of the nerves, but in Victor Pauchet's and Duverger's methods, as exploited by Sherwood Dunn, it is injected at the point of origin of the nerve (nerve block), or along the trunk near the origin, so that the whole region supplied by the nerve and its branches is anesthetized.

In true infiltration anesthesia, the anesthetic effect is obtained more by the pressure of the water and the ischemia than by the weak solutions of cocain. Its effect is instantaneous, while for regional anesthesia by nerve blocking by cocain, procain, novocain, butyn or other local anesthetics, the full effect is not obtained until the drug has acted for thirty minutes to three quarters of an hour.

We owe to Loewenstein of Prague, and to the Greens of San Francisco, the proper tract for anesthetization of the eyeball, that through the lower lid, (and this also absolutely anesthetizes the nasal wall, the sphenoid, ethmoids and frontal sinuses of the same side), without edema or untoward accidents. Pauchet and Loewenstein inject in the upper and inner angle which is sometimes, as I have found it, followed by extensive edema and temporary blindness, even though the optic nerve itself is not reached and is not anesthetized.

Strict attention to asepsis goes without saying in either endermatic or nerve block anesthesia. The six cataract and one strabismus cases operated upon under regional anesthesia in Salt Lake City on the 16th inst., by Aaron and Louis Green, had complete anesthesia and no complications, and enthuse one to adopt this form of anesthesia for cataract and other radical operations upon the eyeball and its adnexae.

DR. OLIVER TYDINGS, Chicago, Illinois: I want only to express my appreciation to Dr. Würdemann for the beautiful language he has used in expressing our appreciation of the work of the Drs. Green, in doing the beautiful cataract extraction work by their local anesthesia. I have watched them do this without any pain, and while I am not prepared to follow them all the way, I appreciate their work. I am not prepared to go all the way for this reason—would not a weaker anesthesia do? In my work I use $\frac{1}{2}$ of 1 per cent. novocain and never more than 4 per cent. cocain.

Another point, is there any possible danger? I recall that some years ago, before a meeting of oto-laryngologists, I heard it stated by some man from Washington, D. C., that a number of fatalities had been reported, supposedly as the result of adrenalin about the region of the nose. I do not recall who it was that made the statement, but know it was some operator from Washington, and would like to know whether

any of you gentlemen recall who it was. Some years ago, I had a report from a friend in Columbus, Ohio, of a death following the injection of adrenalin. I injected an ounce into a Scotch terrier weighing about fourteen or fifteen pounds, and it had no injurious effect whatever. I would like to know whether there is any danger in this method? Otherwise, I can only compliment the Drs. Green on their work.

DR. VERNON A. CHAPMAN, Milwaukee, Wisconsin: Just a word regarding the prevention of accidents. It seems to me that the selection of the word "procain" was very unfortunate. It sounds too much like cocain, and I think some other word should be coined to take its place. In the operating room, if you can *see* the word it is not so bad. When I ask for a solution, I always ask the nurse what it is before I inject it. I say, "What is this?" and I get the word "cocain" in answer. I am in the habit of saying, "Spell it," because I often do not know whether they say procain or cocain.

DR. GEORGE HENRY MOORE, Pottsville, Pa.: I have not prepared any notes on the discussion of this paper. Here are my hands. Last February I had a skin eruption and consulted several specialists, with varying diagnosis. Finally, Dr. O'Leary of the Mayo Clinic thought it was apothecin poisoning. He gave me a subcutaneous injection of apothecin and it produced an extensive redness around the point of injection; and we came to the conclusion that I had apothecin or occupational poisoning. I think this is of importance in considering what local anesthetic to use. They are not innocent of untoward effects.

DR. GEORGE W. BOOT, Chicago, Illinois: I just wish to say something about the point brought out by Dr. Tydings regarding the strength of the solution. I have been injecting about three drams of a 1 per cent. solution of novocain, to which has been added 8 or 10 drops of adrenalin. I did a tonsillectomy in this way in a woman of about fifty, with a blood pressure between 250 and 300. Immediately after the operation, the patient went into collapse, with precordial pain, rapid respiration and rapid heart beat. In this case I think the untoward effects were due to adrenalin given to a patient with such high blood pressure. She received an injection of morphin, and in the course of half an hour the alarming symptoms had subsided and she recovered.

I should like to have Dr. Hayden tell us if there is any way that we can tell in advance whether an idiosyncrasy to cocain may be present.

DR. EMIL MAYER, New York, N. Y.: I have a feeling of pride at something accomplished when Dr. Hayden does the honor of analyzing the work of Dr. Skillern, Dr. Sonnenschein and myself. I desire to bring before you something additional to what we have already presented. The cases here analyzed are taken entirely from that report, and if Dr. Hayden will be good enough to make his headings to that effect, it will add to their value and effectiveness. Many of you know that the Trustees and House of Delegates of the American Medical Association, realizing that we had reported this time twenty-seven and at another time twenty-one deaths, so that we have reported forty-eight deaths in two reports, agreed that it was desirable to go into the entire field of medicine in the study of toxicity in local anesthesia. They have asked me to take charge of it, and we have appointed a committee for internal medicine, surgery, genitourinary work, dentistry, etc. We have sent out over five thousand letters to physicians, and we expect to present a report which will, we

hope, be representative. The representative of ophthalmology is Dr. Lamb, and I can now say that so far we have had no fatalities at all and practically no toxic effects in the eye. If any member present has seen any toxic effect of any kind, I shall be pleased to have him let us know, because we wish to have this report comprehensive.

Dr. Moore presented the toxic effect of apothecin. We have several cases of skin disease brought about by novocain, and if Dr. Moore's diagnosis is correct, I hope he will let us know the results and whether apothecin really produced the condition. If you will wait until our committee has made a report, we may be able to make a report about the proper method of administration of these drugs. Undoubtedly whenever a fatality occurs, the doctor is liable to have a suit for malpractice. I am sure if they will take the two reports made by our committee, and hand them to an intelligent lawyer, the doctor will never be tried. This committee shows that these deaths occur much more frequently than is supposed from the literature. Only five cases have been reported, whereas we have reported forty-eight so far. Dr. Macht of Johns Hopkins, of our Committee, has received some 650 replies, and in them some 10 deaths have been reported from novocain and cocain.

As to procain, the Doctor is entirely right, and the man who suggested that name regretted it when his attention was called to its similarity to cocain.

As to adrenalin, I see no objection whatever to it. We have used it and have seen no bad effects. The ampules that have been made by Metz have been prepared as a result of the report of our committee, and no substitution can occur if these are used.

Regarding submucous resection, I think the speaker is in error when he says deaths do not occur. I should say that about one in five thousand operations gives a fatality. I think you will find enough deaths among the submucous cases.

As to the question of idiosyncrasy, it is just this: There is such a thing as susceptibility. In order to ascertain that, we use $\frac{1}{4}$ of the smallest amount of cocain you intend to use in operating. The person who is susceptible will get some symptoms from a very small amount, and that is our indication so far as that is concerned.

As to recommendations, I cannot anticipate the report that is to be made, and can only say that Dr. Hayden is to be thanked for bringing up this subject. We ask your cooperation in this work, that is going to be of value to the American profession and to American medicine.

DR. A. S. GREEN, San Francisco, California, (closing): In our opinion, the point of entrance of the needle as indicated in our paper is anatomically the most desirable and least likely to meet with untoward results. The length of the needle and strength of the solution are important factors in the success of the anesthesia. We at first tried a one percent solution, but soon found this inadequate. In fact it is sometimes necessary to use a four per cent solution in acute glaucoma and for enucleations. The needle must be no less than 4 cm. or more than 5 cm. long, and the solution not weaker than two per cent. It is of the greatest importance to give sufficient time for the anesthetic to act—at least 45 minutes after injection. We employ only the novocain put up in ampules by Metz. Our results with this have been so satisfactory, that we have been unwilling to try anything else.

FURTHER STUDIES OF THE VITREOUS BODIES.

F. PARK LEWIS, M.D., F.A.C.S.

BUFFALO, N. Y.

I am constrained to say, at the beginning of this paper, that it is with a feeling of some diffidence, that I bring before the Academy the results of observations which differ in so marked a degree from the conclusions which have been generally accepted concerning the vitreous body.

If I have obtained any unusual results, they have been due to the fact that I have used animal eyes largely for experimental purposes, not with the idea that the findings would necessarily be reproduced in the human eye, but with the intention of discovering whether the reaction of this peculiar tissue, at the present time so little understood and which is like nothing else in the human body, were in any measure similar in the different forms of animal life,—whether or not it were possible to demonstrate the presence of any organized structure in it, and if so, to learn, if it could be done, what were its peculiarities, what its relationship and what its functions. These experiments have been exceedingly numerous and varied. Vitreous of various animals and birds, as well as that of the human eye, has been treated in the greatest variety of ways. It has been immersed in solutions of potassium iodid, of mercurochrome, of a long list of alkalies and of acids. It has been soaked in oil, and has been treated by the application of heat and of cold. It has been hardened and stained and sectioned. In this process, some facts have been elicited concerning this relatively huge structure and, fragmentary and inconclusive as they may be, they are yet of a sufficiently striking character, to justify us in the assurance, that in this tissue we have a great undeveloped mine of wealth, inviting to its serious consideration the attention of the ophthalmologist.

In these studies, variations have been found peculiar to the special species from which the specimen has been taken. These differences will open up a wide range for future study. Certain elements, however, have been found to be universally present in the vitreous of animals, of birds and men. It is

with these common factors that the present paper will concern itself. I have for many years felt, and in various papers have endeavored to demonstrate, that the great jelly like, formless mass constituting the vitreous body, occupying four-fifths of the globe in man and consisting of at least 90% fluid, can never be adequately studied other than in a medium which is itself fluid. It is self evident, that if a watery substance has its contents withdrawn from it, even though it may have some unsubstantial framework holding it imperfectly together, the abstraction of its contents will leave only a skeleton, and the relative parts of this will be so dislocated and misplaced, that they can give no adequate idea of its original form and purpose. As well might we expect that a juicy fruit, like the watermelon, if it were placed in a hydraulic press, and squeezed into a compass of one tenth of its normal bulk, would still show when placed under the microscope the delicate intercommunicating channels through which the nutrient fluid passes, and by which its vitality is maintained. But even when the vitreous body has been subjected to this hardening and shrinking process, embedded in celloidin or other supporting material, the section made is not without value, in that it shows that there *did* exist a framework—crowded out of shape though it may be—and that it extended through the entire substance of the vitreous body. This framework will be seen to consist of a large number of fibers many of which are curled up tightly in coils.

Two important facts may then be established: First, the existence of a skeleton form throughout the vitreous body, and, second, that many of its fibers possess the important quality of elasticity.

Before, however, we even open the eyeball, its surface should be studied to determine whether the outside is in any distinctive way modified by its contents. Almost immediately after the eye is removed from the orbit, it will be noticed that there will be a slight tendency on the part of the globe to collapse. This is not very marked, and it is not confined to the entire ball. It is on the posterior half of the globe and is at two opposite points. These are not quite the same either in form or in shape. That on the nasal side takes the form of a circle, as though it had been indented by the tip of the finger. This indentation may be removed by a slight pressure on the opposite side of the globe. The anterior half of the globe

is a firmer tissue than is the posterior, the dividing line being at the equator. It is in the softer tissue that the collapse occurs. All that portion of the ball in front of the median line retains its form. It is in the softer back portion that the collapse is found. On a plane passing through the upper part of the optic nerve and the middle of the cornea, is the lower side of this circle which marks the indentation. The fibers of the sclerotic dip out of their course at this line. This is seen with the naked eye, but more easily with the aid of the binocular loupe. The measurements in the several specimens in the human eye examined were about the same, that is to say one cm. in diameter in each direction. On the opposite side of the eyeball, extending vertically in a plane corresponding with the denser anterior part of the globe, the collapse takes the form of a vertical cylinder nearly two cm. in length and $2\frac{1}{2}$ mm. in width. It marks the junction of the two segments of the eyeball.

The importance of this is, that it would seem to indicate *that the underlying vitreous is of different degrees of density, and that the lessened density is under the areas at which the collapse regularly occurs.*

Another very suggestive fact may be readily determined: It has long been known that if the vitreous, when removed from the eye, is held upon the hand, or placed upon a screen, the fluid which is within slowly percolates away. If it be laid upon a flat surface, which is not too warm, it may be fully 20 minutes or half an hour before the fluid contents are so fully discharged that only a small amount of the jelly like tissue is left behind; but it will be noted that the loss of the fluid occurs **slowly**; it does not pour out as would happen if a **bag** of fluid were opened. That fact logically carries with it the inference, that the fluid which was *contained* in this substance must have been held in *containers*; that some of these containers opened on the surface and some did not; and that those that did open on the surface had small openings through which the fluid could pass only in small quantities at a time; or the only other alternative must have been, that the fluid was so thick that it flowed but slowly. As a matter of fact it may be seen at once, that the fluid discharged is *thin* and watery. Now if the vitreous is cut in two, the same fact is observed, the discharge of the fluid is **slow**. From this, four irresistible conclusions must be drawn; first, that there is a

body to the vitreous; second, that it has a fluid content; third, that there are openings on its surface from which this fluid can be discharged; and fourth, that these openings are the orifices of intercommunicating passages, for if they were not all joined together in some way, only that portion of the vitreous would be emptied with which they were connected. It will be further noted, as a fifth observation, that this discharge takes place only when the vitreous is loosened from the retina, thereby opening the mouths of these passageways. There must then be some connection between the vitreous and the tissue surrounding.

Before, however, we can adequately study the structure of the vitreous body, it is worth while to note what takes place after the fresh eyeball has been opened. If the globe be opened carefully, it will be seen that a watery fluid is immediately discharged. This varies in amount according to the species of animal from which the eye is taken. The loss from the human eye is very small, amounting to only 8 or 10 drops. It is greater in the ox, it is abundant in a horse, the quantity in the great globe of the latter amounting to between 1 and 2 cc. I have called this fluid in order to distinguish it the *vitreous-aqueous*, and its purpose seems to be to fill in the open spaces between the vitreous folds, to be described later. It is probably a lymphoid secretion, and also probably is continuous with the intraocular lymph spaces connecting with the optic nerve sheath, and the postlenticular spaces.

When the globe is opened and the entire lenticular system removed from it, it will be found that the vitreous and crystalline lens are so intimately associated at the ciliary border, that they form one continuous lenticular system. In palpating the vitreous with the finger, one is made conscious at once that it is not of equal density throughout; it grows firmer from without toward the crystalline lens, and it gives to the touch a peculiar feeling of resiliency. The fibers from the hyaloid extend into the suspensory ligament, of which, indeed, as Sir William Lister has pointed out, it is largely composed. Now, not only do these fibers extend into the ciliary ligament, but they extend forward between these, well into the anterior capsule of the lens. If this lenticular mass be put into a watery solution, especially if the solution be slightly colored with staining material, the course of these fibers may be traced well over the anterior margin of the lens. If left im-

mersed in distilled water over night, an interesting phenomenon will be observed—much less marked in the human than the bovine eye. It imbibes the fluid until the vitreous body becomes about half again greater than its normal size; but this imbibition is not uniform. The enveloping membrane swells up like a balloon, while the deeper tissues are but little changed. If now it be examined, while still immersed in the fluid, by throwing a ray of light obliquely through it, it will be seen that a space equal to about $1/3$ of its volume occupies a position between the hyaloid membrane and certain definite curved surfaces, which may be seen within it. The position



Fig. 1. Coagulated vitreous of the ox showing natural divisions when separated. Fig. 2. Coagulated vitreous of human eye showing natural subdivisions.

and relationship of these vary according to the species of animal from which they have been taken. In the eye of the ox there are three of these. In the human eye the denser tissue presents a somewhat different form, and a mass consisting of six bundles seems to group together, extending from the posterior capsule nearly to the opening made by the tearing away of the hyaloid membrane around the optic disc. In the eye of the horse there are two unequal vitreous masses, the one large and bulky, the other membranous and spreading like a veil, across, behind and attached to the posterior capsule of the crystalline lens. If now the distilled water be acidulated, or as I have found more effective, if there be added to

it Gilson's mixture in sufficient amount to make a 5% solution, the denser parts of the vitreous substance coagulate and opacify, thereby distinguishing them and bringing them more clearly into view. If mercurochrome be added, the coloring will increase their visibility. The preparation may be left in the solution for a length of time to be determined by the strength of the solution and the degree of opacity desired. The interior parts of the vitreous will begin to coagulate and if placed again in distilled water, before it has become too opaque, the enveloping membrane will retain its transparency, so that the interior parts may be observed. In course of time, the hyaloid will become either broken or dissolved, and the surfaces which we have seen under focal illumination will be found to be distinctive parts of the vitreous bodies, and will separate from each other, following the natural lines of cleavage dividing it into its component parts. Each has distinctive and invariable attachments and relationships. This invariability shows that these divisions cannot be artefacts, but are the normally separate parts of this organism—which I think we may begin to call *an organ*. Two of these are attached marginally around the ciliary region. These vary, but one portion of this seems to be common to all of the animal structures which I have examined. This takes its attachment directly from the center, or near the center, of the posterior portion of the capsule of the crystalline lens. To this it is firmly adherent and seems to form a part of it. It stands up distinctively when the other parts are folded back or to one side. It appears almost like the mane of a horse, and for that reason I have named it that we may distinguish it from the other parts in referring to it as the *mane*, or the *chaite*. The limits of this paper will not permit the detailed description of these structures which I should like to give. They vary, as I have said, according to the species from which they are taken, and each one might occupy the entire time allotted to the subject.

The possible significance of this attachment was indicated in a very remarkable specimen which I had the good fortune to secure. After the vitreous and crystalline lens of the eye of the ox had remained for some time in the coagulating solution, an interesting phenomenon occurred. The hyaloid membrane became ruptured, and there slipped out from the substance of the vitreous a fairly solid plaque. This was firm and dense, and was made up of the larger portion of the pos-

terior capsule of the lens and the contiguous tissue from the vitreous body. It seemed to have an almost equally firm membrane on the posterior surface. Between these two was transparent vitreous substance. The plaque was thicker in the middle, in which can be clearly seen a perfectly curved surface, which is at right angles to the lens.

This specimen I have the pleasure of showing here today. The lower portion of the curved surface seems to have a



Fig. 3. Lenticular system after being immersed in water becomes swollen, and after the hyaloid is destroyed natural divisions are seen.



Fig. 4. A plaque including the posterior capsule of the crystalline lens showing a lentoid formation. It is clearly seen in No. 2, another view in the horizontal plane.

rounded form, as though it completed the lentoid structure. I use the term lentoid, as meaning having the form of a lens without necessarily performing the functions of focusing the light waves. What the significance of this structure is, I am not prepared to say. It does not seem likely that it can be an actual lens, as being a part of a medium having the same index of refraction, it could have no influence focally in changing the direction of the light waves within the substance of which

it is a part. It would appear rather to be due to the contraction of that part of the vitreous shown to be invariably attached to the posterior surface of the lens, which I have designated as the "mane" or "chaite," which had contracted under the influence of the hardening fluids, and which in shrinking in contracting, had produced this exact form of curvature. Any traction exerted upon these structures, which we have seen have within them elastic fibers, and which could produce such a perfectly regular contraction back of the lens capsule, must exert an influence upon its accommodative function. What effect this may be is not the subject of this paper. This is offered merely as additional anatomic fact concerning this important structure.

I have intimated that the vitreous must of necessity consist of organized structural tissue. The first time my attention was definitely called to the fact was on examining the vitreous of a pig. In this, two fine, thread like fibers which were perfectly transparent in the watery medium in which the vitreous was floated except when brought into view by oblique illumination, were found under the slit lamp microscope to be distinctive tubules. They arose one on either side of the crystalline lens, and extended back toward the hyaloid membrane, in which they connected with a larger peripheral canal.

Subsequently, tubules of like character but differently placed were found in the eyes of other animals, as well as in the human eye, but that again opens up a subject too extensive for consideration within the limits of this paper.

The following then are the facts which seem to have been ascertained:

First: That the vitreous is not a simple undifferentiated mass of tissue with slight septa between its several portions, but it consists of at least three different parts; an enveloping membrane, a watery fluid and a denser portion regularly formed and having organized parts. The enveloping membrane is distensible and elastic, as are the fibers of the framework constituting its structures. The vitreous is not of the same degree of density throughout, but grows firmer in consistency as it approaches the posterior portion of the crystalline lens, and possesses a distinct character of elasticity and resiliency, which communicates itself to the touch of the finger. This more dense portion has a coagulable content. In the human eye, as in the eyes of animals, the center portion

of this dense tissue is directly attached to the posterior capsule of the crystalline lens, with the action of which it in all probability has some direct relationship. That these conclusions are well grounded is only recently receiving the support of eminent authorities.

When Prof. Gullstrand, by his skillful management of the slit lamp in controlling a light ray, made it possible to illuminate the interior of the living eye, many European observers, and especially Prof. Vogt of Basel, described tissues which are found to exist in the living vitreous body. In his exceedingly valuable "Atlas of the Slit Lamp Microscopy of the Living Eye," he described the structures which by dextrous illumination are brought into view. It is possible he says to see the framework of the vitreous. "It has a wavy tunic like form and (in aphakia) often extends into the anterior chamber." With finer illumination, added details were made visible. Parts were brought into view that were supposed to be void of structure. In those empty spaces a "finely fibrous and delicately meshed network of structure" was brought into view. Posterior to the crystalline lens and separated from it by a distinct, "optically empty" space is a "thin folded luminous reflecting membrane," and that important as it is, is all.

In spite of these interesting findings, he concluded we are "still at the threshold of this knowledge." That these should be seen at all, even with perfect illumination and under the microscope, shows a high degree of skill and wonderful patience, because that which Prof. Vogt brought into view were transparent substances enclosing even more transparent substances. That which he saw was undoubtedly the envelopes and partitions of the divisions which I have attempted to describe.

The purpose of this paper is to urge that work on the living eye may be supplemented by effective study on the dead eye. It offers added advantage indeed in the possibilities of placing it in all positions under the slit lamp microscope. It enables us to see tissues which, no longer clear, are made visible either by staining or by the slight postmortem changes which they develop. Their relationships remain the same, and we have in this field of research a new and hitherto unused source of study which has in it great possibilities.

One other method has been employed which tends to corroborate the findings embodied in this paper, and which gives support to the conclusions I have attempted to detail. Sir

William Lister, in a notable paper read at an International Congress of Ophthalmology in Washington, D. C., in April of this year, referring to his researches on the vitreous, said that "pathologic conditions again throw light on the matter." When a foreign body has passed through the vitreous, it is not uncommon to find thin planes of fibrous tissue in one or more places. These planes are very surprising. Doubtless they are due to hemorrhages, but one naturally wonders why the hemorrhages should be limited to a definite plane. He concludes that some radial structure of the vitreous has determined the peculiar distribution of the effused blood.

I had intended to consider in this paper the supporting evidence of a destructive vitreous structure from a pathologic viewpoint, but within the limits of this paper that cannot be done. I hope on another occasion to give further observations to those which I have ventured to present to you to-day.

DISCUSSION.

DR. HARRY S. GRADLE, Chicago, Illinois: The facts presented by Dr. Lewis are extremely difficult to discuss by anyone who has not studied the vitreous in the original and ingenious method described by Dr. Lewis. Therefore, the discussion must of necessity be limited to those phases that have come under universal observation. Should further study corroborate and elaborate Dr. Lewis's findings, a complete revision of our existing knowledge of the vitreous, the action of the ciliary body, the mechanism of accommodation, and the formation of the aqueous will be necessary. It will be extremely interesting to see how Hagen's experiments and theories regarding the vitreous formation of the aqueous will fit with the anatomic findings just described by Dr. Lewis.

Mention was made of the retrolental space. This was originally described by Vogt as a distinct, empty space existing between the posterior lens capsule and the anterior limiting membrane of the vitreous. It was shown to be optically empty. This finding was corroborated and elaborated into theoretic importance by Koeppel as well as Erggelet. At the last meeting of the German Ophthalmological Society at Jena, Comberg demonstrated patients in whom the lens had been extracted, so that the anterior vitreous was in plain view. With the higher powers of slit lamp microscopy, he was able to show that the anterior limiting membrane of the vitreous presented a uniform, nonfibrillar structure, and that behind the membrane was a distinct, optically empty space, separating the membrane from the plainly visible fibrillar structure of the anterior vitreous. Inasmuch as the anterior limiting membrane of the vitreous is so nearly homogenous, that under normal circumstances (that is, in nonaphakic eyes), it is practically visible, Comberg believes that the previously described optically empty space lies in the anterior vitreous and not immediately retrolental.

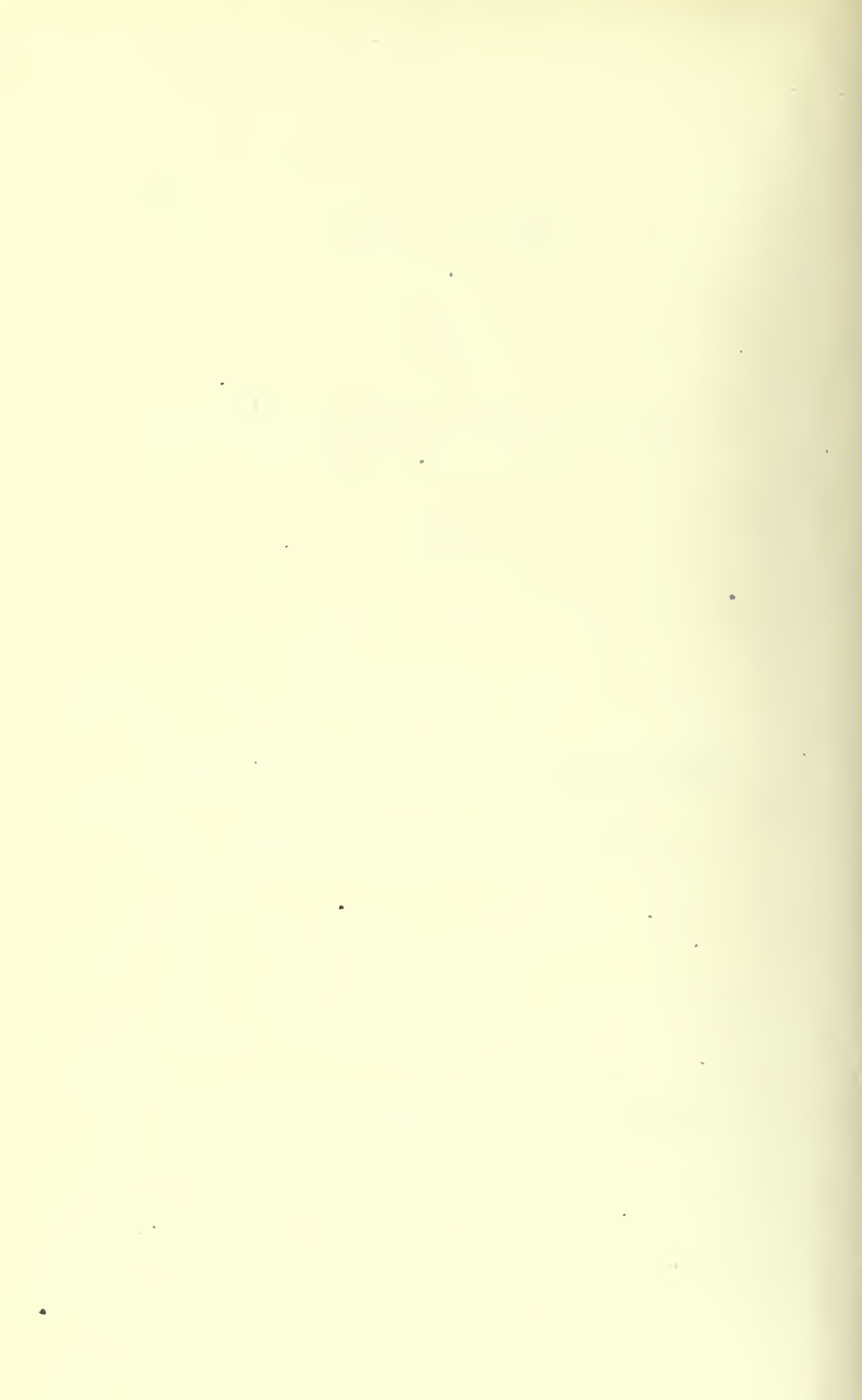
In this connection, an observation that I made but recently may be worth recording. A man suffered a perforating injury by a minute spicule of steel that lodged within the lens and resisted attempts at removal. During the course of six months of close observation, a low degree of siderosis developed, which, together with an increasing opacity, necessitated removal of the lens. At operation it developed that the foreign body had become completely oxydized, and the resultant iron oxid was diffused over and under the capsule of the lens. The operation was successful, and the aphakic eye presented perfect vision with the proper correction. Close observation of the clearly visible anterior vitreous, under 65 diameters magnification with the slit lamp, revealed a unique picture. The anterior vitreous presented a very definite, meshed, fibrillar structure, the interstices of which exceeded the diameter of the fibrils in the ratio of about five to one. No regularity of arrangement of the fibers could be seen. The anterior limiting membrane was practically invisible; that is, it was a homogenous structure that reflected no light from any of its parts. For a distance of perhaps one to one and a half millimeters, in the central part and possibly in the periphery, the vitreous fibrillae were beaded at irregular intervals with minute brown spheres. They looked like hairs covered with nits. I believe that the brown spherelets were minute bits of iron oxid, the pigment of siderosis, that had been carried backward from the lens into the vitreous and become adherent to the vitreous fibrillae. They were not sufficiently dense to interfere with vision, nor were they large enough to be seen with other than high magnification. From their position, the flow of lymph posteriorly from the lens could be predicated, a point that would tend to corroborate some of Dr. Lewis's observations.

DR. F. PARK LEWIS, Buffalo, N. Y., (closing): The further studies I have made have been with the slit lamp microscope. The specimen which has been passed around is a very remarkable one, in which apparently a lens form occurred in the vitreous. What it means I am unable to say.

TRANSACTIONS
OF THE
TWENTY-SEVENTH ANNUAL MEETING
OF THE

American
Academy of Ophthalmology
and Oto-Laryngology

OPHTHALMOLOGIC DIVISION



CORNEAL DEPOSITS OF CHOLESTERIN AND LIME SALTS ENTIRELY DISSOLVED BY THE APPLICATION OF 95 PERCENT ALCOHOL SOLUTION.

LOUIS F. LOVE, M.D.

PHILADELPHIA, PA.

The case here reported is that of Joseph O'Neil, nine years old. In 1908 the patient was treated in the Ophthalmic Department of the University of Pennsylvania, as well as in St. Mary's Hospital, where he applied for eye treatment, giving the following history:

Measles at 3 years of age, since which time the eyes have been inflamed more or less constantly.

The right eye showed an opaque band extending across the lower third of the cornea. Numerous dot like opacities, varying in size and density from pinpoint to pinhead, and in color from gray to chalky white, were present, and were denser and more numerous toward the nasal side. To the outer side the cornea was hazy, and the pupillary space was found to be covered by chalky deposits. V.O.D.=20/50.

The left eye showed opacities that occupied a central position in the cornea; they were circular in outline. The pupillary space was also covered by deposits. V. O. S.=3/100.

A number of the cervical glands were enlarged, but no scars were found. The laboratory reports were inconclusive, and gave a tentative diagnosis of rickets. Night sweats were present.

At that time the clinical diagnosis was uncertain, and rested between keratitis dendritica or the well known band shaped keratitis, or ulcerative keratitis.

In 1916 the patient returned to St. Mary's for glasses. On examination, numerous deposits of cholesterol crystals or lime salts were found distributed over both corneae, including also the pupillary areas. Vision before operation: O.D.=20/70; with correction 20/30; O.S.=15/CC.

Ophthalmoscopic examination of the right eye showed the cornea to be maculated, with numerous deposits of cholesterol or lime salts distributed throughout the structure. No gross pathologic changes in the eyeground were found.

The cornea of the left eye was in practically the same condition as that of the right. In the left eyeground, however, to the

temporal side, a large patch of choroidal atrophy was discernible, with evidences of absorption, surrounded by pigment extending to the macular region. The Wassermann test was negative, as was also the urinary examination. Tubercular tests were not satisfactory.

The patient was admitted to St. Mary's Hospital for operation. The right eye was operated on first. The epithelium of the cornea, and Bowman's membrane which covered the cholesterol deposits were picked off with a discission needle. A 95 percent solution of alcohol was applied. This immediately dissolved the lime, which infiltrated the cornea and spread over the surface, making the entire cornea milky white in color. This was somewhat alarming, and gave rise to the fear that irreparable damage might have been inflicted on the eye. By the following day, however, the solution had been entirely absorbed, and the cornea was clear and bright. After a few days, the left eye was operated on with similar results. The vision improved; O.D., 20/70, with 3 D. \ominus 0.75 D. ax 165 gave 20/20; O.S.=15/200, with S.—3.50 D.=20/100.

A careful search through the literature has failed to disclose the reports of cases similar to the one here described, in which the treatment of cholesterol deposits by alcohol solution was employed. In Wood's *Ophthalmic Therapeutics*, page 394, Birnbacker recommends, for the removal of calcareous deposits in the cornea, that the chalky infiltration be touched with a 5 percent solution of hydrochloric acid, which should at once be neutralized by sodium carbonate of similar strength.

In Noyes' *Diseases of the Eye*, page 382, the following statement is made: "The calcareous deposit occurs beneath the epithelium in irregular specks and lines, and slowly increases during years. It gives rise to no irritation until it attains considerable size and causes erosion of the epithelium; it then acts as a foreign body, and should be scraped away under the influence of cocaine."

In Norris and Oliver, *Textbook of Ophthalmology*, page 346, it is stated that Bowman, Dixon, Nettleship, and other English writers have had good results, and have improved the eyesight by cutting and scraping away the calcareous deposits. In some of these instances the good results have been permanent, and have not been followed by an increase of intra-ocular tension.

J. P. Nuel, of the University of Liege, Belgium, writing in Norris and Oliver's System of Diseases of the Eye, vol. iv, p. 243, states: "Special mention should be made of a rather frequent corneal disease which was described by von Graefe under the title of 'Bandular Keratitis.' On a plane with the palpebral slit, a grayish or somewhat yellowish, nonvascularized inflammation appears, which extends transversely across the cornea; its surface is finely granular or rough, like granite, and rather dry. As for treatment, nothing can be hoped for in clearing up of the macula. If the eye be not amaurotic, scraping of the cornea may be beneficial, as the reformed tissue is ordinarily more transparent than the macula."

In conclusion I feel quite sure that in these cases alcohol is a valuable remedy. What impressed me more than anything else was that our knowledge at the present time as to the chemicophysiologic or pharmacologic action of drugs on the human eye is extremely meager. It seems to me that more numerous experiments and investigations should be made, in order to enlarge our field of physiologic therapeutics. In other words, less attention should be given to the study of pathologic specimens of removed eyeballs, and a more intensive investigation of the physiology of the eye *in situ* should be made.

DISCUSSION

DR. ROY KELLY, Mitchell, So. Dakota: I would like to ask how we can determine whether these deposits are calcareous?

DR. LOUIS F. LOVE, Philadelphia, (closing): In response to Dr. Kelly I would say that we all know that these cases are extremely rare; yet calcareous deposits are readily recognized.

They appear as crystals and are white, grayish white or yellowish white.

NEUROFIBROMA OF THE ORBIT.

EDWARD STIEREN, B.S., M.D., F.A.C.S.

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We have the authority of the President of our Academy, Dr. Walter R. Parker¹, that cases of neurofibromata of the orbit are extremely rare, he being able to find but two cases on record, Terch's and Marchetti's. He quotes Terch as saying in this connection: "A solitary neuroma is altogether a rare tumor, and its location in the orbit appears at least a curiosity." Parker's case made the third to be recorded, which makes it appear that the case about to be reported is the fourth in the literature of this unusual growth in the orbit.

The patient, a Hebrew, age 28, was referred by Dr. H. G. Wertheimer, June 12, 1919, to be refracted. The eyeballs were equal in size and in position in the orbits; pupils were equal and responded normally to light and to accommodation. Muscle balance, 4° esophoria distance, orthophoria near. R. V. 6/20. L. V. 6/5. Under homatropin and cocain:

R. V. = 6/100, +5.50 S. \ominus +0.25 Cyl. ax 75° = 6/8.

L. V. = 6/40, +2.00 S. \ominus +0.25 Cyl. ax 150° = 6/4.

The media were clear and the fundi negative. Both fields were normal for form and color. A comfortable correction was ordered, which was increased to almost full strength three months later. Shortly afterwards, interesting developments began to manifest themselves in and about the right eye. The left eye can be ignored as it has remained practically the same.

October 27, 1919, he appeared, complaining of a full feeling in the right orbit and malar region. The vision in the right eye without correction was found to be reduced to 2/20. Under homatropin cycloplegia, he was again refracted, retinoscopy revealing 7.50 D. hypermetropia, and the trial lens improving vision to 6/15+ only. The retinal veins seemed slightly larger than in the left eye, but no distinct pathology in the fundus could be detected. He was referred to Dr. George J. McKee for sinus examination, but all accessory sinuses were found to be normal.

A stronger correction was ordered for the right eye, which he wore with comfort. He was lost sight of for a year, when he appeared with a definite feeling of fullness about the right



Fig. 1. Before operation.

orbit, and it was seen that the right eye was becoming more prominent than the left. Under homatropin, the error of refraction in the right eye had increased to +10 D. Sph., which gave vision of only 6/30. The retinal veins had become extremely tortuous with a certain amount of retinal edema, mainly in the lower part of the fundus. The field of vision, which heretofore had been normal for form and colors, showed a marked reduction for red, 5° above and 10° below, while the field for green and blue amounted to central fixation only.

Blood and spinal Wassermanns, X-ray of the teeth and sinuses all proved negative. A diagnosis of tumor of the orbit was then made, probably involving the optic nerve. He was exhibited before the Pittsburgh Ophthalmological Society, October 25, 1920, and all the members present concurred in the diagnosis. Potassium iodid was exhibited over a period of six months, and the eye kept under close observation. Exophthalmos developed very slowly. In fact, as the photograph shows, exophthalmus was never very prominent. November 30, 1920; limitation in movement of the eyeball upwards was noted, and ocular movements in all directions became more restricted as time progressed.

Operation was urged, but as no assurance of the outcome could be given, it being thought that the tumor involved the optic nerve, the patient would not accede until annoying diplopia became constant. Desiring an outside opinion, a consultation was arranged with Dr. George E. de Schweinitz, who wrote in abstract as follows: "It seems to me that the indications are definitely those of a tumor in the posterior portion of the orbit, and almost certainly connected with the optic nerve. The extraordinary edema of the optic nerve and edema of the retina, fully 10 D. in height, with the huge twisting of the vessels, etc., all seem to me more or less characteristic of an optic nerve growth, and it is distinctly my impression that surgical interference is indicated." On his return the patient consented to an operation, and accordingly was admitted to the Presbyterian Hospital, May 17th, 1921.

Having performed the Kroenlein operation some five or six times on the cadavar, I have come to regard it as a most difficult procedure in gaining a route to the posterior part of the orbit. I wish to subscribe to everything Edward Jackson said in his discussion², on Byers excellent papers on Tumors of the Optic nerve³, at Atlantic City in 1914, especially that Kroenlein's operation has a very limited field in ophthalmology, and that in many cases of exophthalmos the posterior

pole of the eye is on a level with the outer margin of the orbit. The configuration of the patient's head showed that he must have a shallow orbit with a broad base, and it was decided not to resect the outer wall.

After the usual preparation and under ether, a horizontal incision was made, beginning at the external canthus and carried for about three inches toward the temple. After the outer periosteum was retracted, a half-inch of the outer orbital rim was removed with chisel and rongeur. A horizontal incision in the periosteum of the orbit was made above and



Fig. 2. Actual size of tumor.

parallel to the external rectus. Through this opening a finger was introduced, and a round, hard mass below and behind the globe was felt. It was not attached to the optic nerve, but arose from the floor of the orbit, well back, and was removed by finger dissection. It was encapsulated, slightly nodular and almost round, measuring 25 mm. in diameter and weighing 5.5 grams. After the slight hemorrhage from the orbit had been checked by gauze sponges, the incision in the periosteum was sutured with No. 00 chromic catgut, the outer periosteum replaced, and the wound in the soft parts approximated with

silk worm sutures. Healing was uneventful and the patient left the hospital on the sixth day after the operation.

The specimen was given to Dr. de Wayne G. Richey, Pathologist to Mercy Hospital, for microscopic study, who conferred with Dr. Samuel R. Haythorne, Professor of Pathology in the University of Pittsburgh, on the unusual and rare type of tumor it proved to be. Their description follows, and I desire to take this opportunity of expressing my deepest gratitude to them for their careful and painstaking study of the sections.

LABORATORY REPORT

"Sections of the tumor mass from the floor of the orbit showed a cellular structure covered by a fibrous capsule. The tumor consisted, for the most part, of wavy, parallel or intertwining bundles of fibrous connective tissue. These varied considerably in their density and cellularity. As a rule, the tumor tissue was compact, the fibrous fasciculi being arranged quite closely together. Where they were looser, irregular empty spaces were seen, but these were not associated with mucoid or myxomatous degeneration, nor could any areas of necrosis be seen. The tumor cells were elongated and of a spindle type when cut longitudinally. Their cytoplasm was homogeneous. The nuclei were long and fusiform, finely granular and devoid of mitotic figures. Where a whorly arrangement was encountered, so that the cells were cut obliquely or in transection, the tumor appeared quite cellular and the cut ends of the cells had a round or oval shape, with oval vesicular nuclei. Here and there small crescentic clusters of cells were seen, which did not suggest the rosettes of a neuroblastoma as described by Wright. In the sections examined, no nerve tissue could be demonstrated. The tumor was not unduly vascular, there being a moderate number of well formed blood channels scattered throughout it. Where the structure was most cellular, a finely granular, golden pigment, presumably blood pigment, occurred in the interstices between the cells. A few lymphocytes and plasma cells were scattered diffusely through the sections.

Sections stained by phosphotungstic acid hematoxylin showed, at the periphery of the tumor, a moderate amount of collagen, resting in the intercellular spaces. The more central portions of the tumor were made up of younger connective tissue cells, some of which were definitely fibroblastic in nature. An occasional fibril could be demonstrated, but none of these could be identified as neuroglial fibrils.

Diagnosis: It is clear that this tumor is a fibroma. In the orbit, fibromata derive their origin, most commonly, from two sources—the periosteum or the nerve trunks. The general structure of this tumor consists of numerous bundles of fibrils, which are not extremely heavy, which show a marked tendency to intertwine, and between which fusiform cells often lie. As Ewing⁴ indicates, a diagnosis of neurofibroma can be safely made on such findings, calling attention to the fact that, in periosteal fibromas, the bundles of fibrils are much larger and

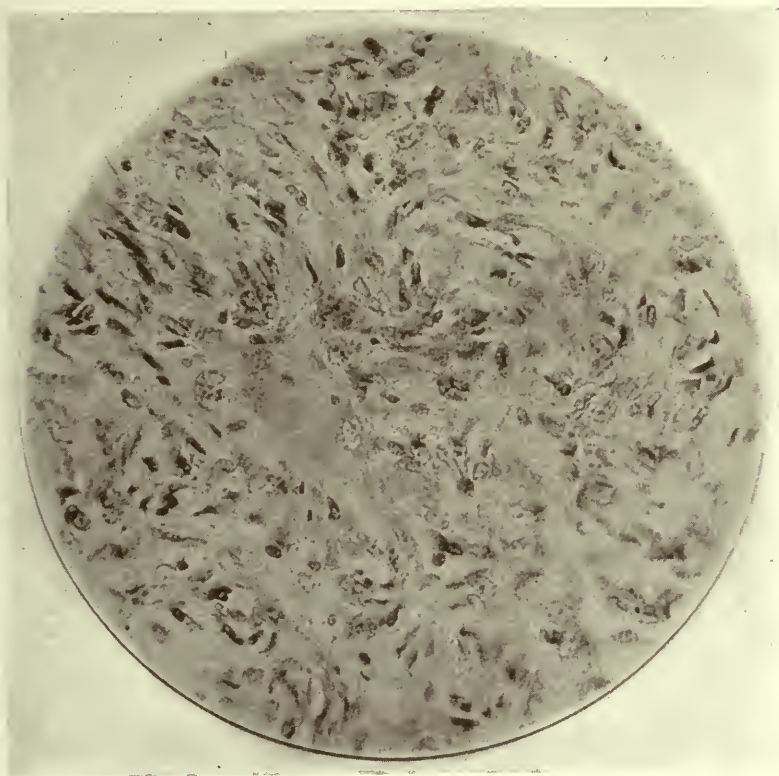


Fig. 3. Photomicrograph of section of neurofibroma of the orbit.

the intertwining is not so outstanding. It is also known that in the fibrous types of neurofibromata, nerve fibrils are usually missing, so that their demonstration is not essential for the diagnosis. There is also the probability that the fibromas can arise solely from the supporting tissues of the nerve, the nerve cells not taking part in the proliferation. In this instance, the diagnosis is neurofibroma, indicating the source of the tumor even in the absence of nerve tissue."

It has been interesting to observe the behavior of the eye



Fig. 4. After operation.

following the operation. As the photograph illustrates, there is now a condition of enophthalmos, but this is scarcely noticeable behind the strong plus lens worn. The scar has healed until it is barely visible. The fundus picture has likewise entirely changed; the edema has disappeared, but there is and will always remain some tortuosity of the veins. The false hypermetropia has receded and the lens now accepted, +6.50 S., gives vision of 6/10—. Excursion of the eye is satisfactory in all directions excepting on extreme outward movement, where there is some lagging, due no doubt to adhesions to the scar in the periosteum.

Conclusions: I. Progressive increase in hypermetropia does not necessarily indicate tumor of the optic nerve, nor does edema of the retina and optic nerve. Either condition may develop from any steady pressure from behind.

2. Operative measures should be instituted early to preserve the retina and optic nerve. Bull in his excellent monograph⁵ states, "There may be an artificial hypermetropia by flattening of the eyeball from pressure of an orbital tumor. At first the ophthalmoscopic picture is negative, but as the pressure from behind continues and the obstruction to the return circulation becomes more complete, the retinal veins become engorged, the arteries narrowed and the picture resembles finally that of a papillitis, with hemorrhages in the retina and more or less extensive retinitis, ending in atrophy of the optic nerve."

3. Single neuromata of the orbit are unique and rare. They are not to be confused with multiple fibroma, where one or more nodules may form on the lids, the lobe of the ear and in other remote localities. Bull⁶ makes no mention of neurofibromata of the orbit. He merely mentions fibroma of the orbit, and suggests Berlin's view that they should be classed as fibrosarcomata.

4. Orbital tumors can many times be removed without recourse to the Kroenlein operation. A horizontal incision in the temple and removing part of the rim of the orbit (if found necessary) is a much easier procedure and gives better cosmetic results.

REFERENCES.

1. Parker, Neurofibroma of the Orbit. Kroenlein Operation. Trans. Sec. Oph. A. M. A., 1907.
2. Trans. Sec. Oph. A. M. A., 1914, p. 44.
3. Ibid.
4. Loc. cit.
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TUMORS ARISING NEAR THE APEX OF THE ORBIT.

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Tumors and cysts of the orbit, arising in the neighborhood of the optic foramen, may originate in the soft parts of the sphenoidal fissure, near the apex, in the optic nerve or its sheath, or in the soft tissue of the orbit itself. Very rarely, a tumor of the bone or a cholesterin cyst will arise from the walls of the orbit.

Tumors and cysts arising from bony tissue are usually slow growing and produce changes that can be detected by means of the roentgenograms. A shadow will be cast by a thin bony contour protruding into the orbit, or as a result of bone destruction. Soft tumors rarely give rise to changes discernible in the roentgenogram, even though the normal content of the orbit is entirely extruded, and there is extensive destruction of soft tissues from the rapid growth of a malignant neoplasm.

Small tumors may exist in the posterior part of the orbit for years, growing very little and only slightly interfering with ocular rotation and vision. The earliest symptom in such cases is protrusion of the globe, or proptosis. This may precede visual disturbances for several years, and disturbance of motility for several months, depending on the size of the tumor.

Next to proptosis, edema of the lids is the most common symptom. Often the swelling of the lids, with little proptosis and no palpable evidence of orbital tumor, has been thought to be due to nephritis or sinus disease. It is rather uncommon to find a person with swollen lids associated with orbital tumor, who has not had an intranasal operation for relief of that symptom. Swelling of the lids is usually greatest when the tumor overrides the globe in the superior and nasal quadrants; this sign may serve to indicate the most probable location of the tumor.

The operation of choice for the removal of a retrobulbar tumor depends largely on the appearance of the lids and the position of the globe, except in instances in which the tumor can be definitely palpated beneath the rim of the orbit. Hudson advises

removal of tumors of the optic nerve through the soft tissues, unless some vision remains, or there is some doubt with regard to the diagnosis. If there is vision, he advises the Krönlein operation. Even though an eye is sightless, it is well to try to remove the tumor without enucleation and without destruction of the globe.

In tumors of the soft tissues, I prefer to use the direct frontal route, making a skin incision as for a Killian operation. The soft parts should be cut down to the bone, about 0.6 cm. above the superior orbital rim. The periosteum should be elevated around the margin of the orbit, and the periorbita on the superior and nasal sides, and the contents of the orbit depressed and retracted until a finger can be easily inserted almost to the apex. The orbital contents may then be palpated and even a small tumor felt anywhere within the orbit. The periorbita should then be incised nearest the location of the tumor, and the mass removed by blunt dissection with small scissors or forceps, care being taken to mutilate the orbital structures as little as possible. The Krönlein operation is distinctly valuable in removing tumors within the muscle cone, when it is desired to save the globe. The contour of the face and orbit, however, often renders the operation quite difficult, as the lateral wall of the orbit may be quite thick, and, when turned back, allows little additional room for work.

Tumors of the intracranial portion of the optic nerve are rare, as are tumors of the optic nerve and its sheath. When they can be diagnosed, the operation described by Dandy may be advisable. Hudson, in 1912, collected 154 cases of tumor of the optic nerve, and I have been able to collect reports of forty-six other cases from available literature, although some of these are evidently repeated cases, and in some the diagnosis was not confirmed. I wish to present here the clinical data and the pathologic reports on eight cases I have observed of tumor or cyst arising from the optic nerve or its sheath.

REPORT OF CASES.

CASE 1 (A366379). Miss V. B., aged eighteen years, entered the Clinic July 27, 1921, because of proptosis and swelling of the lids of the left eye (Fig. 1). In September, 1920, she first noticed transient swelling of the left eyelids, which later became continuous and marked. Pain had not been associated with this swelling until a month before, when she had had a feeling of pressure through the temples, more marked on the left side. She

had not had diplopia. The vision was not impaired, and the extraocular movements were normal.

Examination.—Vision in both eyes was 6/5. There was some swelling of the lids of the left eye and proptosis of 8 mm. The globe was pushed directly forward. In both eyes the media were somewhat hyperemic, with slight blurring of the margins of the disc. The retinal vessels were normal. A roentgenogram of the head revealed definite increased density over the area of the



Fig. 1 (Case A366379). Photograph showing proptosis of left eye nine months after onset. Tumor removed by direct frontal route.

left orbit. Examination of the nose and pharynx was negative. A complete general and neurologic examination was negative, except for a compensated cardiac lesion of long standing. A diagnosis of orbital tumor was made by exclusion, and exploratory operation was advised.

Operation.—August 10, 1921, an incision was made along the rim of the orbit from the middle of the brow to below the external canthus, through the palpebral ligament. The periosteum was

excised, and dissection carried down to the apex of the orbit. A soft mass at the apex, which seemed to be connected with the lacrimal gland, could not be entirely removed, but after dissection was carried through the cone to the nerve, pieces of the tumor could be removed with forceps.

Microscopic examination revealed endothelioma. A section contained numerous medium sized, oval and round vesicular nuclei (Fig. 2), between which was a fair amount of fibrillar intracellular material. The blood supply was generous, and the larger vessels had well formed walls. The nuclei varied in size, but stained uniformly; mitotic figures were not conspicuous.

The patient was dismissed September 1, after radium had been applied. The media of the left eye were clear, but there

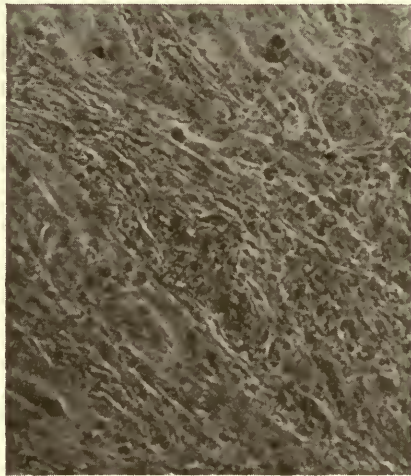


Fig. 2 (Case A366379). Section of endothelioma (x 200).

was 2 diopters of swelling of the nervehead, and the retina was edematous. The vessels were engorged and tortuous, but no hemorrhages were visible.

February 27, 1922, the patient returned for examination. She had had considerable continuous headache for the past three months, from the reaction produced by the radium. During the last two or three weeks, the headaches had been less severe. Vomiting did not occur except on the day after radium had been applied. The swelling in the left orbit never receded after the removal of the tumor. A few weeks after the patient returned home, the sight of the right eye failed rather rapidly, so that for the past three weeks vision had amounted to only light perception. The vision of the left eye, which was good when she left

the Clinic, had also failed rapidly. On examination, she had no light perception in the right eye, and only light perception and doubtful projection in the left eye. There was moderate edema of the lids of the left eye and proptosis. The exophthalmometer readings were 24 left and 17 right. External ocular movements of the left eye were markedly impaired, but the patient could move the eye for a slight distance in all directions. The movements of the right eye were not impaired. Examination of the right eye showed the cornea to be clear, and the anterior chamber of normal depth. The pupil was dilated to about 6 mm. in diameter, did not react to direct light, but did react to consensual stimulation. The media were clear. The nervehead was swollen about 4 diopters. The retina around the nervehead for 2 or 3 disc diameters was irregularly swollen. The veins were engorged and extremely tortuous. There were wide areas of white exudate along the course of all the vessels for a distance of 2 disc diameters from the disc. The macular region was thickly spotted with white dots and lines, giving the appearance of an imperfect star. The cornea of the left eye was clear, and the anterior chamber of normal depth. The pupil was dilated to about 4 to 5 mm., and reacted to direct light. The media were clear. The nervehead was swollen to about 4 diopters. There was swelling of the retina for 2 disc diameters beyond the disc margin, with intense engorgement of the veins. There were areas of white exudate along the line of some of the vessels and marked inflammatory changes in the region of the macula, similar to those in the right eye. The patient had slight buzzing and ringing in both ears and pronounced impairment of hearing in the left ear.

A few weeks later, an exploratory operation was performed elsewhere. A large infiltrating tumor was found to extend along the base of the brain from the optic foramen back as far as could be seen. The pathologic diagnosis on tissue removed from the brain was endothelioma.

CASE 2 (A311349). Mr. B. P., aged twenty years, entered the Clinic in April, 1920, because of prominence of the left eye. Seven years and seven months before, he had fallen from a bicycle, striking on the back of his head; he was unconscious for one-half hour. He vomited frequently for twenty-four hours. Nineteen months later, he had mumps, and immediately afterward, his mother noticed slight proptosis of the left eye, which had increased gradually for two years. Since then (about four years) the condition had remained practically stationary. In April, following the onset of proptosis, the vision of the left eye

began to diminish. In August, he was told by an oculist that the optic nerve was inflamed. Vision was 6/60 and had remained stationary for the last three years. Every summer for four years, he had had an attack of severe pain, beginning in the left



Fig. 3 (Case A311349). Photograph showing proptosis of left eye six years after onset. Note the contracture of the fingers of the left hand, due to neurofibroma in elbow. Tumor removed by Krönlein operation.

eye and passing to the right eye. This was followed by vomiting of short duration.

Examination.—The upper and lower lids of the left eye were swollen and of soft, doughy consistency. The lids were deeper red than those of the right eye. There was a ptosis of 4 mm., due

to edema, and 9 mm. exophthalmos directly forward by Hertel's exophthalmometer. In the primary position the visual axes were parallel. There was good movement of the left eye to the right and to the left, although there was some limitation of movement in the field of the external rectus. The eye could be elevated only about 10 degrees and depressed about 20 degrees. In the depth of the fornix, the vessels were injected. No definite mass could be palpated. The orbital margin was not eroded. The eye could not be pressed backwards into the orbit. There was no



Fig. 4 (Case A311349). Section of tumor with optic nerve going through the mass.

swelling of the face beyond the orbital rim. The cornea was clear, the iris dark blue, and the pupil round, 2.5 mm. in diameter, equal to the right pupil. The light reflex was present but diminished; the convergence reflex was good. Vision equalled 6/60. There was papilledema of 2 diopters. A small tumor removed from the left anticubilateral fossa for diagnosis proved to be neurofibroma. A small tumor removed later from the leg also proved to be neurofibroma. A tumor from the spinal cord was diagnosed endothelioma (psammoma).

Operation:—A Krönlein operation was performed April 20, 1920. A firm, fibrous, flat, round tumor, 2.5 by 1.2 cm. in diameter, was found near the apex of the orbit, with the optic nerve running through its center (Fig. 4). The mass was separated from all other structures within the muscle cone and removed, including a short section of the optic nerve. The eyeball subsequently shrank and was removed some months later. Ptosis became complete and the swelling of the lids did not recede. The patient has had no recurrence of the tumor.

A microscopic diagnosis of endothelioma, (psammoma) was made. A section of the specimen contained groups of cells with small, round, or oval nuclei and abundant, finely granular cyto-

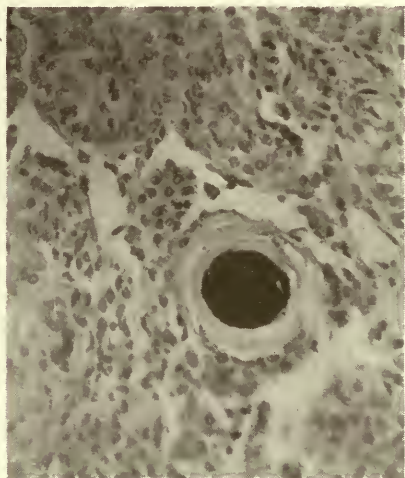


Fig. 5 (Case A311349). Section of endothelioma showing psammoma body (x 200).

plasm (Fig. 5). These nests of cells were separated by bands of well developed fibrous tissue of varying width. In some places, the fibrous tissue was as abundant as the cellular areas. The cross section of the optic nerve was included in one section. The tumor cells surrounded the nerve and were found in the outer layers of the sheath, but did not penetrate it. Scattered through the same section were round bodies, large enough to be seen with the naked eye; these stained deeply with hematoxylin. In the anilin blue stain, they were seen to be made up of concentric layers of a hyalin like substance, psammoma bodies.

CASE 3 (A323522). Miss A. M., aged thirty-four years, entered the Clinic July 7, 1920, because of a mass in the right orbit

behind the upper lid (figs. 6 and 7). The first sign of swelling around the orbit occurred seven years before, and had advanced without pain. Six years before, vision began to fail in the right eye, and the failure had been gradual. Since 1916, there had been no noticeable change in the proptosis. An aspiration was attempted during the first year of the swelling, but no fluid was obtained. No history of trauma, or of serious illness could be obtained.



Fig. 6 (Case A323522). Photograph showing proptosis of right eye seven years after onset. Tumor removed by direct frontal route.

Examination.—A solid mass was found under the right upper lid, below the brow, which bulged far forward, pushing the eye downward, 10 to 12 mm., and inward. The mass was about 35 mm. by 27 mm. The skin of the upper lid was freely movable. It extended toward the nasal side only to the superior orbital notch. The conjunctiva was not adherent, and was normal in appearance around the upper fornix. Movement of the right eye

was impaired upward and laterally, not nasally or downward. There was no noticeable erosion of the orbital rim. The media of the eye were clear, the discs small, and margins blurred. There was slight proliferation of the nerve fiber sheaths above and below the disc following the course of the larger vessels. The optic cup was shallow, the lamina cribrosa was not visible; the nerve was pale pink. The fundus was otherwise normal.

Operation.—July 13, 1920, an incision was made through the middle of the upper lid parallel with the brow. The tissues were divided down to the tumor, which lay outside the ocular muscle



Fig. 7 (Case A323522). Photograph showing position of the right eye five months after operation.

cone. The tumor, 4 by 3.5 by 3 cm., was nodular, round, smooth, and completely encapsulated, with the base rather firmly attached to the sphenoidal fissure. There were no blood vessels communicating with it, except at the base of the sphenoidal fissure. On dissection, the tumor was found to be composed of a rather firm bunch of white, spongy nodules. Figure 7 shows the position of the right eye five months after operation.

The gross specimen measured 35 by 28 by 20 mm. It was firm, generally oval in outline, with several small nodules projecting from one pole. The tumor was definitely encapsulated, and the cut surface showed prominent fibrous trabeculae.

A microscopic diagnosis of endothelioma was made. The section contained groups of rather small cells separated by a delicate network. The nuclei of these cells were oval, fairly regular in size, shape, and staining, but in general were rounder, slightly larger and lighter staining near the centers of these groups (Fig. 8). In the center of some of the groups, the cytoplasm had an opaque, hyalin appearance and the nuclei stained faintly. In general, the cells were arranged concentrically; in some places they formed definite whorls, in others they were flattened and lined small cavities of varying sizes, which suggested blood vessels.

CASE 4 (A369116). Mr. T. L. M., aged forty-five years, en-

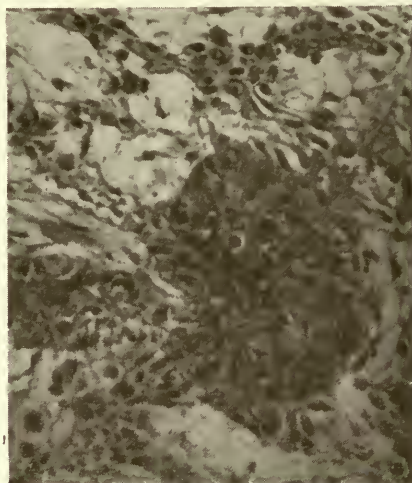


Fig. 8 (Case A323522). Section of endothelioma (x 200).

tered the Clinic August 18, 1921, because of proptosis of the right eye and pain in the head. He had first noticed pain in and over the right eye about ten months before. The pain was neuralgic in type, starting in the right eye and shooting backward on the right side of the head. Three months after the onset of pain he first noticed protrusion of the right eye; this had increased gradually. The pain had decreased during the past few weeks. The eye had been slightly inflamed at times, but only for short periods. For ten years, he had noticed deafness in the left ear with a ringing and roaring noise. He had staggered somewhat when walking, but this was thought by his relatives to be due to pain, and the bent over position he assumed. Increasing loss of memory had been noted during the last two months. Recently, he had com-

plained of slight headache in the left occipital and left frontal regions.

Examination.—Vision of the right eye was 6/7, of the left 6/6. The eyes were normal in shape and size, but the right eye protruded. The exophthalmometer readings were right eye 24, left eye 18. There was a slight edema of the right lids but none of the conjunctiva. The right internal rectus muscle was weak, but otherwise the ocular muscles were unimpaired. The right cornea was clear, the pupil was 4 mm. in diameter and reacted normally. Intraocular tension was normal. The pupil dilated freely with cocain, the media were clear, the disc was round, and the margins were blurred. The cup was obliterated, and slight edema of the retina was noted. There was a large retinal hemorrhage on the nasal side of the disc, extending one-half a disc diameter beyond the disc margin. There were a few punctate hemorrhages scattered over the fundus. The retinal veins were slightly engorged and tortuous. The media of the left eye were clear, the disc was round and margins were blurred. The cup was shallow with lamina cribrosa obscured. There was a fairly large retinal hemorrhage under the superior temporal vein, extending one-half disc diameter beyond the disc, and a small linear hemorrhage on the upper nasal disc margin. There was slight edema of the retina, no change in the vessels, but a few small punctate hemorrhages were scattered throughout the fundus, particularly on the temporal side. No pathologic lesions could be found in the nose or antrum, although a slight fullness was present in the right lower lid and upper cheek. This was carefully investigated because of a previous diagnosis of malignant growth of the antrum extending into the orbit. Roentgenograms of the head were reported to be negative. The neurologic findings were indefinite. The patient was very dull, attention rapidly wandered, and he showed poor comprehension. Any test requiring cooperation was badly performed. The presence of an orbital tumor seemed evident, with a recent "slump in intelligence," which suggested invasion of the frontal lobe. In order to clear up the diagnosis, the orbit was explored.

Operation.—August 28, 1921, an incision was made along the superior nasal margin of the orbit. The periosteum was elevated to the apex of the orbit on the superior and nasal sides. A boggy mass could then be palpated inside the muscle cone. The priorbita was opened on the superior sur-

face, and the tumor found lying along the side of the optic nerve and extending from the globe backward almost to the apex of the orbit. The tumor was rather firm, movable, and could be separated from the optic nerve only with difficulty. It was about 1 by 2.5 by 2 cm.

A microscopic diagnosis of endothelioma was made. The section (Fig. 9) was made up of large, lightly staining cells, with large vesicular nuclei and abundant cytoplasm. The nuclei varied greatly in size; there were numerous mitotic and many multinucleated cells. There were several large areas of round cell infiltration, and the tumor cells of these areas were degenerating and necrotic. In places, the tumor cells were arranged in cords.

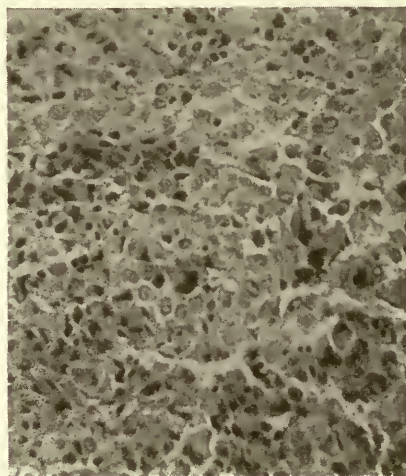


Fig. 9 (Case A369116). Section of endothelioma (x 200).

The patient did not convalesce well. He showed increasing stupor, disorientation, and other signs of frontal lobe tumor. October 3, an exploratory decompression was performed, with osteoplastic flap of the right frontoparietal area, and the tumor, which extended up into the lobe, was encountered on the under surface of the frontal lobe. In view of the position and extent of the lesion, a radical operation was not considered advisable. The patient died January 3, 1922.

CASE 5 (A-354470). Mr. E. F. P., aged thirty-four years, entered the Clinic, August 5, 1921, because of protrusion of the left eye of two years' duration. The vision in this eye, however, commenced to fail in 1911, and the eye had been

totally blind since 1919. The day before the onset of pain, the patient had drunk some port wine, which he thinks may have contained wood alcohol. Pain in the orbit was increased by drinking any alcoholic beverage or by eating sour food. A cold seemed to make the eye protrude more than usual. The patient had been treated for toxic amblyopia and for sinuitis. In January, 1920, the vision of the right eye became blurred for three weeks, then recovered. Just before he entered the Clinic, he again had a period during which vision in the right eye was blurred. As the blurring was more noticeable for close work, he attributed it to the action of hyoscyamus in the laxative pills he was taking.

Examination.—General and neurologic examinations were made, but failed to reveal a pathologic condition, aside from that presented in the left orbit. The right eye was normal. An extensive investigation for evidence of syphilis was also negative. Vision, with glasses, was 6/5 in the right eye; in the left eye nil. There was considerable proptosis of the left eye, which was directed outward and somewhat upward, but measurements were not recorded. Movements of the left eye were limited in all directions, but particularly in the field of action of the internal rectus. No palpable mass could be found around the orbital rim. The tissues of the lids and globe were normal to external appearance. The pupil was 6 mm. in diameter, did not react to direct light but contracted promptly to consensual stimulation and on attempt to converge. The media were clear. The disc was oval, the long axis was vertical and pearly white, with evidence of former papilledema. There was slight exaggeration of the choroidal ring on the temporal side, the cup was obliterated, and the lamina cribrosa was not visible. The nasal side of the disc was raised and blurred. There were a number of finely tortuous vessels on the nasal portion, and on the inferior temporal quadrant of the disc. The disc was not measurably elevated over the lower part of the surrounding retina, but was raised 1 diopter in the nasal portion, above the upper temporal portion of the retina. The veins were slightly tortuous. The inferior and nasal veins showed well marked periphlebitis for a distance from the disc, as did also the inferior temporal, but less markedly so. There was perivasculitis along the branches of the nasal arteries, and less well marked perivascular changes were evident along the other veins. The choroid, in general, showed exaggeration

of the interspaces and some degenerative changes in the pigment layer. This was particularly noticeable in the macular region, where there was a well defined pigment migration. A roentgenogram of the head revealed cloudiness in the left frontal sinus, but was otherwise negative.

Operation.—April 20, 1921, an incision was made below the brow, through all tissues to the muscle cone. Exploration of the orbit was made with the finger and revealed a soft tumor about the size of the globe, surrounding the nerve and lying mostly within the muscle cone. As it was impossible to remove the tumor without sacrificing the eye, the eye was then enucleated and the tumor, which was the size of the

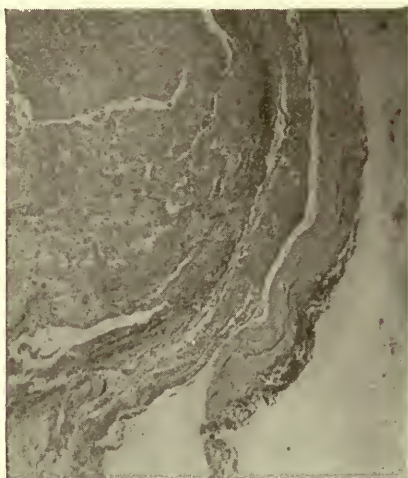


Fig. 10 (Case A354470). Degenerating gliomatous cyst showing the firm capsule (x 18).

globe and soft, with a degenerating central mass, was removed. The outer portion contained a dense capsule, bluish-gray, and very firm, while the central portion was gray, soft, granular, and fragile. The optic nerve passed through the center of the tumor. There was no connection between it and the outer walls.

The gross specimen was a round, definitely encapsulated mass about 2 cm. in diameter. The thick capsule resembled the sclera, and the mass was similar to the eyeball in size and shape. The contents were soft.

A microscopic diagnosis of glioma was made. The sections were stained with hematoxylin and eosin, Mallory's anilin blue connective tissue stain, and Mallory's phospho-

tungstic acid hematoxylin. Along one side of the section was a dense connective tissue capsule (Fig. 10), which resembled the sheath of the optic nerve, with the arachnoid and pial layers exaggerated in places. Beneath this, were numerous large blood vessels and slight infiltration of the tissue with blood corpuscles. In this area, were numerous phagocytic cells distended with brownish granules and blood pigment (rare in a tumor of this kind). The main part of the section was made up of a network of coarse and fine fibrils, which stained deeply with phosphotungstic acid hematoxylin, but did not stain with the anilin blue. In the meshes of this network were scattered cells, whose nuclei varied greatly in size and

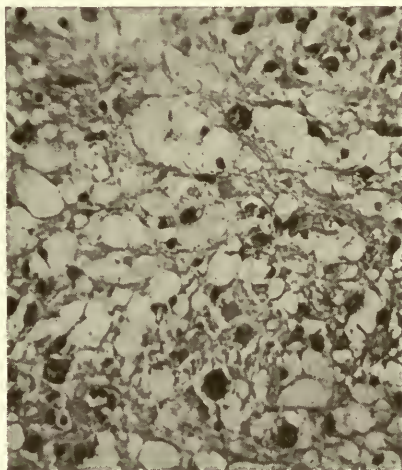


Fig. 11 (Case A354470). Central portion of degenerating gliomatous cyst (x 200).

staining. Some were very small, others fairly large, and the intensity of the stain seemed to have no definite relation to the size (Fig. 11). There were a few cells which appeared to be multinucleated, probably phagocytic endothelial leucocytes. The tissue had a vacuolated appearance (like liver in fatty infiltration), and in some places there were larger spaces partially filled with clear, light staining material. No evidence of tumor was found in a section of the eyeball.

CASE 6 (A399476). A. B., a girl aged seven years, was examined at the Clinic July 27, 1922, because of marked protrusion of the right eye (Fig. 12). This was first noticed in December, 1921. Considerable proptosis appeared in a single night, unaccompanied by pain, and complete blindness

in the eye ensued within a few days. The proptosis reached its height within a month, and for six months had remained about stationary.

Examination.—The general physical examination was negative, and all tests for syphilis were negative. There was no vision in the right eye; in the left it was 6/10. The right



Fig. 12 (Case A399476). Photograph showing proptosis of right eye eight months after onset. Tumor removed by Krönlein operation with preservation of the globe.

eye was directed slightly outward and downward. Upward rotation was limited; the eye could be raised only slightly above the primary position; otherwise rotation was good. The eye could not be pushed back into the orbit, and no mass could be palpated around the rim of the orbit. The media were clear, the disc was round, and the margins were well defined and very pale. There was no loss of nerve substance;

the lamina cribrosa was not visible. Otherwise, the fundus findings were negative in both eyes.

Operation (Krönlein).—July 31, 1922. A soft mass could be felt through the periorbita behind the globe. The periorbita was incised above the external rectus, which was de-



Fig. 13A.

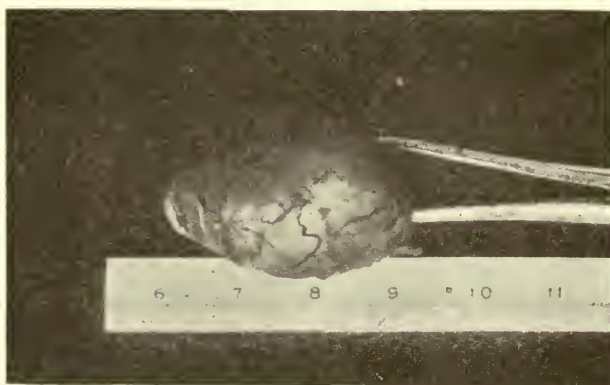


Fig. 13B.

Fig. 13 (Case A399476). (A and B) Photograph showing the size of the tumor. The end grasped by the forceps was the nearest the globe. Note the optic nerve protruding from the tumor.

pressed without being cut. Tenon's capsule was opened over the tumor, parallel with the course of the optic nerve, and the encapsulated tumor found to extend from the apex of the orbit to the globe, tapering at both ends. The greatest diameter of the tumor was about 12 mm., behind the globe.

The nerve was cut immediately behind the globe, and at the apex of the orbit after the tumor had been freed. There were no fibrous adhesions except near the anterior end. The tumor, 32 by 18 by 17 mm., was smooth and rather soft, white, and practically bloodless within; on removal very little hemorrhage occurred. Recovery was rapid and uncomplicated. The eye was retained with good movement and normal appearance.

The gross specimen was pale, pinkish, smooth, and pear-shaped (Fig. 13A). It was fairly firm, but fluctuated slightly. The posterior end, which lay in the apex of the orbit, was 6 by 7 mm. About 7 mm. from the anterior pole was a depression, on the under surface, in which rose the

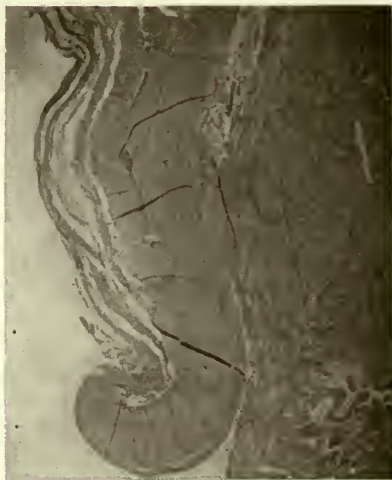


Fig. 14 (Case A399476). Glioma of optic nerve showing the end of the nerve nearest the globe (x 8).

stalk, which was attached to the posterior pole of the eyeball, probably the optic nerve (Fig. 13B). With the exception of this depression and the cut posterior end, the mass was covered by a smooth capsule. A longitudinal section of the tumor was made in the vertical plane, through the optic nerve. The capsule, which is 0.5 to 1 mm. thick, was attached only by a few fine threads, and was wrinkled as though it had been distended by fluid. Along the lower border of the mass was a band, 5 mm. wide, which was continuous on the anterior surface with the optic nerve in the depression on the under side. The anterior half of this band was free; from the upper margin of the posterior half, there apparently arose the main

tumor. The entire cut section was firm, white or yellowish, and appeared to be composed of the same tissue.

A microscopic diagnosis of glioma of the optic nerve was made. Sections were stained with hematoxylin and eosin, Van Gieson's and Bielschowsky's silver stain. The dural, arachnoid and pial layers of the optic nerve sheath were clearly seen on the lower edge of the section, but the two latter were absent or inconspicuous above (Fig. 14). The anterior free end of the band along the lower border, previously described, was undoubtedly optic nerve, but it very soon changed character and resembled the remainder of the section. There was rather a rapid transition from nerve tissue

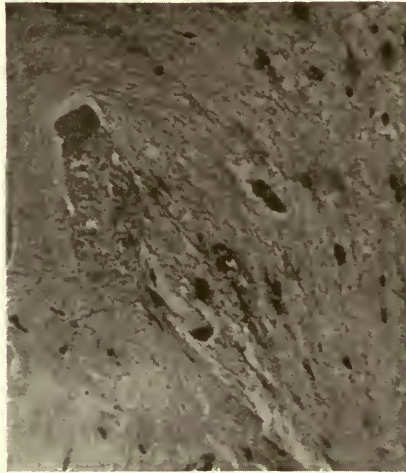


Fig. 15 (Case A399476). Glioma of optic nerve showing the invasion of the optic nerve ($\times 200$).

to tumor tissue. The course of the nerve through the tumor could be surmised by the parallel arrangement of the connective tissue septa. The entire section was made up of a fine network of fibers, in which were numerous cells with oval nuclei (Fig. 15). The fibers did not stain red with Van Gieson's stain, except a few coarser ones around the blood vessels; they stained beautifully with silver. The nuclei varied considerably in size and staining reaction, and mitotic figures seemed to be present. The proportion of cells to fibers varied; and, in general, the more numerous the cells, the larger the nuclei. Much of the tissue was distinctly vacuolated, and in one place, where the cells were smaller and more regular,

they were arranged around these vacuoles as though lining them. There may have been one or several layers of nuclei. Often the vacuoles apparently coalesced to form larger spaces, which contained clear material.

CASE 7 (A382408). Mrs. G., aged thirty years, came to



Fig. 16 (Case A382408). Photograph showing proptosis of left eye twelve months after onset. Tumor removed by direct frontal route.

the Clinic January 24, 1922, because of failing vision and increasing exophthalmos of the left eye (Fig. 16). In September, 1920, she had an acute swelling of the left upper jaw, with infection around her left upper wisdom tooth. The left side of the face was swollen, the left eye had protruded, and caused severe shooting pains. An attempt was made to extract the

tooth and establish drainage, but the roots were broken off and left in place. Within a week, the eye receded considerably, but remained noticeably prominent. It had pained occasionally, but not so severely as at the onset of the swelling. In January, 1922, the patient noticed that vision in the left eye was not so good as it had been. The roots of the broken tooth were removed, three other teeth extracted, and tonsillectomy was performed. She was also given electric treatments. These operations, however, had no effect on the eye or the vision. She still suffered slight pain in the sun in the early morning or late evening. During the week preceding her first examination at the Clinic, the pain in the eye had become infrequent and mild, but she had frontal headache and a dull aching sensation in the right eye. Pain seemed to radiate to a spot on the right temple that was sore to touch. There was also a tender spot in the top of the head.

Examination.—Vision in the right eye was 6/5; in the left 1/60. The lids closed and opened normally. There was proptosis of the left eye, the exophthalmometer reading was 16 in the right eye and 23 in the left eye, the intraocular tension was estimated by palpation to be normal. The external examination was otherwise negative. The pupils were equal and reacted normally. The ocular movements were not impaired. The fundus of the right eye was normal. In the left eye there was an edematous swelling of the nervehead reaching 2 diopters, without hemorrhage or exudate. The details of the disc were obscured by the swelling; the arteries were not affected, but the veins showed moderate distention and tortuosity. The periphery of the fundus was normal. A roentgenogram of the head showed the paranasal sinuses to be greatly enlarged. The nose contained a quantity of pus streaming from the right ethmoidal region. The left side did not contain pus. Antrum puncture on the left side was negative. Exploration of the anterior ethmoids on the left, and probing into the left frontal sinus failed to show lesions.

Operation.—February 7, 1922, brow incision as for a Killian operation was made, and the roof of the orbit explored. Through the periorbita, back of the globe, a soft mass could be palpated, which appeared to be about 1 by 2 cm. This was lying between the periorbita above the optic nerve on the mesial side and the muscle cone on the temporal side. On being freed from its adhesions, there was a gush of clear

fluid containing yellow and white flocculent bodies. The sac was then found to extend to the orbital foramen, where it was tied off by catgut and the accessible portion removed.

The gross specimen presents the collapsed wall of a cyst, which was probably 25 by 15 mm. when distended. The wall was tough but not very thick, and it appeared to be made up of white fibrous tissue.

Microscopic diagnosis of glioma was made. Section showed an oval, cellular mass about 4 by 5 mm. It consisted of parallel rows of closely packed, small, spindle shaped nuclei surrounding groups of smaller round or oval nuclei (probably the same type cut at different angles) (Fig. 17). There was

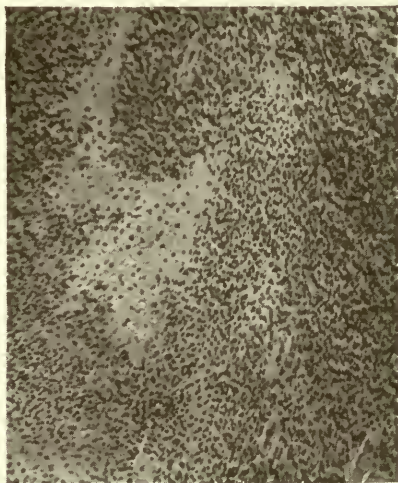


Fig. 17 (Case A382408). Degenerating gliomatous cyst (x 100).

a moderate amount of lighter staining material, made up of closely packed, fine fibers, which were abundant in spots and separated the nuclei widely. The nuclei were fairly uniform in size and staining; no mitotic figures were seen.

CASE 8 (A350846). R. S., a boy, aged eight years, entered the Clinic February 26, 1921, complaining of diplopia and protrusion of the left eye. Diplopia first appeared about two months before, and he was given glasses. No other trouble was noticed at that time. One month later, the parents noticed that the left eye was becoming more prominent. The local physician kept him under observation for one month, then referred him to the Clinic for further examination and treatment.

The patient was an only child, was delivered with instruments, and at birth weighed $9\frac{3}{4}$ pounds. A thin, linear scar running along the left temple, by the outer canthus, over onto the malar prominence, showed the injury that had resulted from the use of forceps. The child was robust, and had been in good health since birth. He had had no headache, and with the diplopia and exophthalmos, had only occasional stinging pains in the orbit and shooting pains over the head. He had vomited several times in one day, five weeks before, but the vomiting was not projectile and was associated with fever.

Examination.—The right eye was normal in shape and position and rotated to normal limits. The left eye showed marked proptosis and was rotated upward until the lower margin of the cornea appeared in the middle of the interpalpebral zone. There was complete paralysis of all the depressors of the left eye and weakness of the left external rectus. The lids were not swollen, but the conjunctival vessels were dilated and tortuous. The cornea was clear. The pupils were equal and normal in size, shape, and position, and reacted normally to light stimulation. Deep palpation around the margin of the orbit failed to reveal a definite mass. The globe could not be pushed back into the orbit. Ophthalmoscopic examination showed the media to be clear. There was a papilledema of 6 diopters, with some swelling of the retina, but no large hemorrhages or exudate. A roentgenogram of the left orbit was reported to be negative. Ears, nose and throat were normal.

Operation.—March 2, 1921, the orbit was opened by Krönlein's method under general anesthesia. A tumor was found in the depth of the orbit, extending forward almost as far as the lower lid, involving the muscle cone and in contact with the optic nerve. At the apex of the orbit, the nerve was surrounded by a soft mass. The tumor was not definitely encapsulated, only slightly vascular, and quite friable. It was removed piecemeal.

A frozen section made at the time was reported as probably sarcomatous by the pathologist, and a subtotal exenteration of the orbit was performed. Radium was applied as follows: March 3, 1921, 1800 mg. hours; March 24, 1000 mg. hours; July 1, 2200 mg. hours; and September 17, 1020 mg. hours. With the exception of a small amount applied in the

center of the orbit, the radium was applied over the left temporal region, screened by 2 mm. of lead and 2.5 cm. of wood. The orbit has since become epithelized, and one year later there was no sign of recurrence.

The gross specimen was a funnel shaped mass about the size to fit into the orbit, and was composed of fatty tissue and firmer nodules. At the apex of the funnel was a round opening, through which the optic nerve probably passed. The globe, with 8 mm. of optic nerve, was preserved separately.

A microscopic diagnosis of neurocytoma, probably neuroblastoma, was made. Sections were stained with toluidin blue, Van Gieson, hematoxylin and eosin, Mallory's anilin

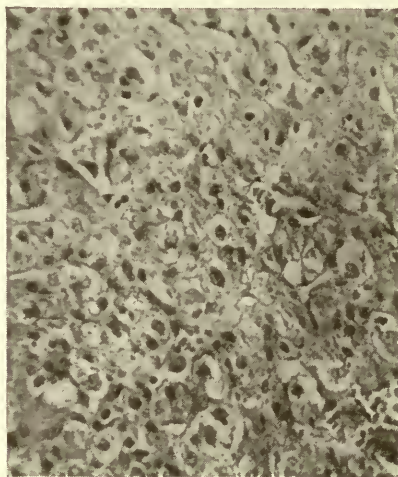


Fig. 18 (Case A350846). Section of neurocytoma (x 200).

blue, Mallory's phosphotungstic acid hematoxylin and Bielschowsky's silver stain. The sections were very cellular and contained very little stroma (Fig. 18). The most conspicuous cells had large light staining oval nuclei with a deeply staining nucleolus in each. Toluidin blue did not demonstrate Nissl bodies. In the silver stain, an occasional cell with processes could be seen. Between the cells were fine fibers, which tended to a concentric arrangement with respect to the cells; this made the section appear as though it was made up of numerous small round units.

The child was alive and well eighteen months after the operation.

SUMMARY OF CASES.

Case	Orbit	Vision in Affected Eye	Proptosis	Duration of Proptosis	Lateral Displacement	Restriction of Ocular Movements	Fundus	Operation	Pathologic Diagnosis	Results
A366379	Left	5/5	8 mm.	Nine months	None	Negative	Direct frontal	Endothelioma	Brain tumor
A311349	Left	6/30, failing seven years	9 mm.	Six years	Out, up, down	Papilledema, 2 diopters	Krönlein	Endothelioma, psammoma	Eye removed eight months later
A323522	Right	6/30, failing six years	Seven years	In, down 12 mm.	Out, up	Slight pallor of disc	Direct frontal	Endothelioma	Good
A396116	Right	6/7, failing ten months	Ten months	Out, 6 mm.	In	Papilledema, 1 diopter, retinal hemorrhages	Direct frontal	Endothelioma	Brain tumor, died five months later
A354470	Left	None, eight years	6 mm.	Two years	Out, up	In out, up, down	Postneuritic atrophy	Enucleation and removal of tumor	Glioma	Good
A399476	Right	None, eight months	8 mm.	Eight months	Down 1 cm.	None	Simple atrophy	Krönlein with preservation of globe	Glioma	Good
A382408	Left	1/60, twelve months	7 mm.	Twelve months	None	Papilledema, 2 diopters	Direct frontal	Glioma	Good
A350846	Left	6/60, two months	Two months	Out, up 7 mm.	Out, down	Papilledema, 6 diopters	Krönlein evisceration	Neurocytoma	Good

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A CASE OF MALIGNANT LYMPHOMA OF THE LACRIMAL GLAND.

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BUFFALO, N. Y.

Tumors of the lacrimal gland are uncommon, so that the report of isolated cases are warranted. In Volume 5, No. 6, June, 1922, American Journal of Ophthalmology, there is a painstaking study of the Tumors of the Lacrimal Gland by Laura A. Lane of Minneapolis, in which there is a historic review, summary of the embryology, and histology of the lacrimal gland, a discussion of the pathology of its tumors, and classification of the types of new growths reported.

As Dr. Lane has pointed out, only 229 cases have been reported in 323 years. A study of the cases made in her paper shows, contrary to general belief, lacrimal tumors are not benign, showing a death rate of 12.63 percent, and that metastasis is to be reckoned with.

CASE REPORT.

Mrs. J. E. H., age 51 years, first seen July 1920. Family history without interest.

The patient contracted syphilis from her husband 18 years ago, for which she underwent thorough treatment from competent medical advisors over a period of five years, and has had frequent examinations, with no evidences of syphilis since her primary infection. Eight months ago, a small, hard, firm lump appeared in the left tear gland region, which has gradually continued to enlarge. She consulted a competent ophthalmologist who, in view of her specific history, put her under a thorough course of mercury, without influence upon the growth of the mass. There has been no loss of weight or reduction in her general well being.

Examination. The patient is a woman of spare frame, but appears wiry and strong. An examination made by her family physician reveals no general pathologic condition; the kidneys, heart and circulation are normal. There is no enlargements of the lymph nodes. Repeated Wassermann tests, submitted to several laboratories, are negative. One spinal puncture reported as negative.

Right vision 6/15, corrects to 6/5—1; left vision 6/15+1,

corrects to 6/6—3+2. Neither fundi show ophthalmoscopic changes, there being no changes in the retinal circulation in either eye. There is a slight ptosis on the left side, which is not increased upon the instillation of cocain. Excursion of the globe is free in all directions, except some impairment in extreme upward excursion. Exophthalmos of L. compared with the right eye, measured by the Hertel instrument. The pupil reactions are normal.

In the lacrimal gland area, there is an oblong mass, parallel to the orbital rim, measuring approximately 18x10 millimeters. The skin is freely moveable. A furrow can be made out, which corresponds to the orbital rim, but the mass, which is quite hard, is apparently firmly lodged in the fossa. Upon eversion of the lid, a mass presents behind the tarsal plate. The preauricular glands are not palpable, nor is there enlargement of the sublingual. Stereoscopic plates of the head showed no thickening of the orbital bones.

Under infiltration anesthesia at the Buffalo General Hospital, through an incision parallel to the orbital margin, a mass was found in the lacrimal fossa, which was fairly firmly adherent to the periosteum. The fossa was cleaned out and a mass, measuring 25x15x8, removed. There was no capsule or trace of gland structure. The wound closed kindly and promptly.

The specimen was sent to the State Institute for the Study of Malignant Diseases, from which the following report was made: "Specimen removed from the eye of Mrs. J. E. H. shows same to be of neoplastic character. The growth consists of round cells, with deeply staining vesicular nuclei and very scanty protoplasm. The cells are closely packed and are supported by a coarse connective tissue stroma running through the structure, with only a slightly reticulated stroma between them. In some portions of the growth, there is a slight suggestion of alveolar arrangement. Thin walled capillaries are found running through the substance of the growth. The structure, on the whole, closely resembles that of a lymph node, without the special structure characteristic of these organs, and presents a picture of malignant lymphoma."

Within a few days after leaving the hospital, the patient was referred to Dr. B. F. Schreiner, who subjected the patient to heavy doses of radium emanations.

There has been no recurrence, or change in the movement of the superior rectus or levator in the two years since the operation.

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BILATERAL TUMORS OF THE LACRIMAL AND PAROTID GLANDS; MIKULICZ SYNDROME.

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REPORT OF CASE.

The case which I propose to report has proven to be one of extreme interest to me from a number of angles, and I am hoping that it may be worth while to you who have a much greater experience than I, and a much wider field for observation.

I would suggest that the greatest interest perhaps centers in the questions of: (1) Rarity, (2) Diagnosis, (3) Treatment.

I have added the subtitle "Mikulicz Syndrome," employing it, however, with very much the same feeling that I have when discussing "Orthodoxy" in religion: I am not quite sure that I could give an acceptable definition of Orthodoxy, neither am I certain that I will be able to convince any body but myself that mine is a case of "Mikulicz." Perhaps it may relieve embarrassment later on, if I confess now that I am not sure that I know what Mikulicz Syndrome actually is, but I have been making an honest effort to find out.

On May 18, 1922, William Malone, col., male, age 18, presented himself, and the examination was as follows:

Family History: Father living, good health; mother, died six years ago, cause unknown; brothers, two living, both older than patient and in good health; sisters, two living, both older than patient, good health, one dead, cause unknown.

I may say in passing that it is difficult to get a definite history of any sort from this race, especially those who are illiterate as this man was.

Personal History: Negative, except for attack of measles three years ago. Two years ago had some swelling in the left eye for two or three days.

Physical Examination: Patient is a vigorous looking, well developed farm hand. Both parotid glands show hard, infiltrated, immovable masses, which slowly increased in size until they got as large as my fist. There is a visible and palpable tumor in each orbit, occupying the upper and outer quadrant, apparently consisting of two portions, presenting distinctly differ-

ent features. The superior one, about the location of the lacrimal gland, feels like cartilage, and is slightly movable beneath the rim of the orbit; the inferior portion, about the location of the accessory lacrimal gland, projects beneath the tarsal cartilage when the lid is everted, is soft, freely movable and apparently vascular. Except for the drooping of the lids and the presence of the mass in each orbit, there was no abnormality, either in or



Fig. 1



Fig. 2

about the eyes. Tissues normal, eye movements normal, pupil reaction normal, tension normal, vision normal, fundi normal.

Examination of the throat, nose and accessory sinuses by inspection, transillumination and X-ray negative.

Careful physical examination by a competent internist revealed no abnormality. This examination was made with special reference to the question of leucemia, but there was no discoverable involvement of the spleen, or other ductless glands, or of

any lymphatics anywhere. No salivary glands other than the parotids were involved.

Urinalysis negative. Blood picture normal:

Red cells 4,920,000, white cells 5,000, neutrophiles 68, eosinophiles 1, basophiles 0, large and small lymphocytes 27, large mononuclears 4, hemoglobin 85%, color index 0.86; Wassermann, blood and spinal fluid negative.

Injection of old tuberculin for diagnostic purposes negative. Dr. Kerlin was much interested in the diagnosis of this case and took great pains with the tuberculin test. He reported no reaction, either constitutionally or locally. It was, therefore, impossible to substantiate either past or present syphilis, tuberculosis or leucemia.

On July 5th, under general anesthesia, the left orbit was operated. The tumor mass removed, hard as cartilage, was roughly 2 inches long, 1 inch wide and $\frac{1}{2}$ inch thick. It was firmly embedded and reached nearly to the apex of the orbit, one rounded smooth end projecting anteriorly. The mass was firmly embedded of course, but not attached to the periosteum. It apparently involved both the lacrimal and accessory lacrimal glands.

The tumor was prepared for microscopic examination, a piece being sent to the Army Medical Museum, at Washington; Major Coupal, Curator, took great interest in the case and sent a slide to Dr. Verhoeff for examination, which he was kind enough to make. His report in detail will follow.

The most interesting feature of the case developed when the reports began to come in from a number of well known pathologists, whose opinions were solicited. They include the following: (1) Blastomycosis, (2) tuberculosis, (3) adenofibroma, (4) mixed cell tumor, (5) No definite pathologic diagnosis.

Dr. Verhoeff reported as follows:

"The specimen consisted of a lacrimal gland. The acini are greatly reduced in number, and those remaining show various stages of atrophy. They are being replaced by fibrous tissue. The stroma is greatly increased in amount. Scattered throughout the gland are many tubercles, most of them miliary in size. These consist of epithelioid cells and giant cells, and are almost free from round cell infiltration. In the larger tubercles, especially, the epithelioid cells have become stellate and have formed mucin. There is no necrosis. The stroma, especially about the comparatively normal acini, is considerably infiltrated with plasma cells. Blood vessels are few in number and some of them show endarter-

itis. The inflammatory process is suggestive of tuberculosis or syphilis, but is not typical of either.

It would be well to carry out animal inoculations in this case."

Dr. John A. Langford, of New Orleans, reported as follows:

"A careful study of this slide shows that it is definitely an inflammatory reaction, which is characterized by formation of tubercles, and it therefore falls in the group of 'infectious granuloma.' I can quite definitely rule out blastomycosis, fibroadenoma and mixed cell tumors, and since the only other known conditions which will produce such reaction are syphilis and tuberculosis, it is most probably one of these two. Since the blood and spinal fluid examinations have proven negative for syphilis, we have narrowed down the condition to tuberculosis. But this is a most unusual manifestation of tuberculosis infection and therefore causes me to be a bit uncertain."

Dr. Jno. H. Dunnington, New York City, says:

"A careful examination of the tissue by the pathologist at the New York Eye and Ear Infirmary shows it to be a fibroadenoma of the lacrimal gland. It also shows myxomatous changes and round cell infiltration. He does not consider it malignant."

Dr. Kenneth M. Lynch, Dallas, Texas, was kind enough to examine the slides and gave his opinion in substance as follows:

"The eye specimen is in my opinion an infectious granuloma on the order of blastomycosis, although I have not seen any organism of that sort in it. It is an infection of apparently lacrimal gland tissue, and seems to have been a duct infection, producing degeneration of gland tissue with granulation and giant cell formation, somewhat on the tubercle order. However, there is vascularization and organization instead of necrosis, and I do not think it is tuberculosis. Look for round yeast or mold like bodies on the order of blastomycosis. I am reasonably certain that it is a fungus infection."

Dr. MacCallum, of Johns Hopkins, very considerably studied the slides and made some valuable suggestions, in part as follows:

"I think this is an instance of Mikulicz disease, as suggested, but it seems that hardly any two cases of that disease show exactly the same histologic characters. A great many of them have shown accumulations of lymphoid tissue, rarely with giant cells, and associated sometimes with changes in the blood, so that the suggestion of a relationship with Hodgkin's disease or with leucemia has been made. At other times a granulation tissue with giant cells has been found, and search for tubercle bacilli or

something similar has been made. Still others have suggested a relationship with syphilis. In this particular case, the lacrimal gland is widely spread apart by a granulation tissue, partly made up of nodules very like tubercles, with giant cells, epithelioid cells and a loose reticulum, together with a great many lymphoid cells. I think, of course, you should stain a lot of sections for tubercle bacilli and perhaps, if possible, for spirochetes or for any other bacteria, but I have a suspicion that you will not find them easily, if at all. If any more tissue is removed, it ought to be inoculated into a guinea pig or several pigs. This has been done in some of the other cases which were less like tuberculosis, but without results."

Drs. Ellis and Butler, of the Shreveport Laboratories, submit the following opinion:

"Examination of several sections shows a chronic form of inflammatory growth, in which a nodulated granulation tissue, together with giant cells, some epithelioid cells and many lymphoid cells are present, and tend to separate the lacrimal gland. There is a formation of fibrous connective tissue also. We do not consider it tuberculous or syphilitic. It is not malignant. We think it is a mixed tumor of the lacrimal gland. Sections were also examined for the spirocheta of syphilis, and for tubercle bacilli, and were negative for both.* Guinea pig inoculation was negative for tuberculosis."

I began the study of this case with the hope that it might result in fixing a little more definitely the exact pathology of Mikulicz' Syndrome, and in order to give the study authoritative standing, the slides were sent to a number of pathologists of recognized authority with request for an opinion. Some of these, because of their unusual interest have been quoted in detail. To all of them I desire to express my thanks and appreciation for the painstaking study which they have made, and the cheerfulness with which they have responded to my request for help.

I fear I must admit, however, after all, that the conclusions in a recent article, to which I shall refer later on, are correct to date, viz: "(1) That Mikulicz is not a separate disease but a syndrome; (2) That the etiology is not due to any one known cause."

1. *Rarity.* It has been difficult to gather any reliable statistical information as to the frequency of Mikulicz, but my impression is that it is rather a rare occurrence. I am hoping that the discussion may prove or disprove that assumption.

In 1909, Howard¹ gave a detailed study of 55 cases accumulating over a period of approximately 21 years. It is estimated that there have been reported since that time about 70 others, making a total of about 125 cases contained in the literature of 35 years; of course there may have been a great many unreported cases. Ophthalmic Literature refers to 11 articles on the subject during the year ending July, 1921. I have been able to find very few references since that time.

2. *Diagnosis.* Since Mikulicz made his first report, in 1888, of what he considered to be a new disease, there has been a good deal of confusion in the writing and thinking on the subject, and as a matter of fact still is, especially as to the identity, exact pathology and etiology.

His original case seems to have been one of bilateral swelling of the lacrimal and parotid glands, without discoverable lymphatic or blood changes. If this strict classification be adhered to, then the condition may be conceived of as an entity, otherwise it would seem to be a syndrome. But it is difficult to stick to this strict classification, and as a matter of fact has not been done. Some cases have been reported as Mikulicz, which at the time showed either other lymphatic involvement, or such blood changes as would seem to relate them closely to the leucemias, or developed them later; some terminating fatally as such. On the other hand, lacrimal and parotid enlargement are known to be a phase of leucemia.

Dr. C. Fisher says² "A continuous series may be demonstrated of all combinations from isolated swelling of the lacrimal glands to general pseudoleucemia, and it may be added lymphatic leucemia as well."

There was published in the June number, A. J. O., an article by Dr. Lane³, of Minneapolis, entitled: "A Study of Tumors of the Lacrimal Gland with report of a Mixed Tumor," which includes a thorough study of Mikulicz, and a comprehensive review of the literature up to July, 1921.

I am quoting freely from this article, and at the same time acknowledge Dr. Lane's great personal kindness in helping a study of my own case with suggestions, and help in reviewing the literature of the subject.

One conclusion from her study is, "Four types of Mikulicz are described in the literature.

"Type 1. Symmetric enlargement of the lacrimal and one or more pair of the salivary glands. There are no blood changes in this type, nor is there any evidence of lymphatic disturbance. 37

out of 59 cases studied showed no changes other than the glandular enlargement.

"Type 2. Symmetric enlargement of the same type as 1. A reduction in the hemoglobin but blood otherwise normal. Some lymphatic enlargement present. The picture is that of pseudo-leucemia.

"Type 3. Symmetric enlargement of the lacrimals and salivary, together with enlargement of all the lymphatics, and in addition the spleen and liver. Profound blood changes with great leucocytosis. Rapidly and progressively fatal, a true leucemia. 22 of 59 cases studied were found to belong to types 2 and 3.

"Type 4. Frenkel declares that 1% of all the so-called enlargements of the lacrimals in Mikulicz' syndrome are physiologic, such as the enlargement due to grief and weeping, menstruation, lactation. Lagrange and others have reported cases which confirm Frenkel's views.

A benign condition, with only lacrimal enlargement, may pass from type 1 to type 3, and death occur within two months."

Lindgren⁴ reports such a case, showing "a hard, indolent swelling of both tear glands, and a corresponding swelling of all the salivary glands, especially the parotid. Besides, all palpable lymph glands showed some swelling. Spleen and liver normal. Anemia increased steadily, swelling of the spleen and liver appeared. Death followed in 7 weeks."

Thursfield⁵ remarks: "It would appear quite clear, that the disease originally described by Mikulicz is, if not the same, at least very closely allied to the affection which involves the lymphatic tissue, and that in turn is very closely related to the leucemias."

All of which is very closely related to the mooted point as to whether the condition is one of direct infection, or a local manifestation of a general systemic disease. Mikulicz thought that his original case was due to direct infection, but he did not identify the infective agent. Local infections have been associated with some cases, but have not been proven to have a causal relation.

Igersheimer and Pollot⁶ concluded, after reviewing 55 articles, that tuberculosis is rarely if ever a cause. Detzel⁷, on the other hand, after studying 43 cases, concludes that T.B. is a frequent cause. Tubercle bacilli have been found in the tissue of some cases reported as Mikulicz.

It is interesting that several of the reports on the tissue from my case raise the question of probable tuberculosis, possibly due

to the presence of giant cells. One pathologist gave it as his opinion that the tissue was the result of tuberculosis. As stated before, however, it has been impossible to find T.B. in the tissue, and the animal inoculation was negative.

3. *Treatment.* This patient has been in the hospital and under constant observation and treatment for about four months. The result of the treatment has been very gratifying.

As stated above, the left orbit was operated on July 5th, with



Fig. 3



Fig. 4

the result that the parts on that side soon resumed a normal appearance and function, except for a slight scar along the edge of the eyebrow, there being at this time neither ptosis nor impaired action of the extraocular muscles. For some weeks after entering the hospital, the parotid masses were slowly but appreciably increasing in size; they were considered inoperable. The patient got ten drops of iodid of potash, sat. sol., three times a day from that time on. About the same time, therapeutic

doses of X-ray were begun as follows: Nine inch distance, 3 mm. aluminum filter, 7 minutes exposure with 4 one-half milliamperes, spark gap seven to eight inches, and applied to the left parotid gland only, that is, the parotid on the same side as the operated orbit. He had about six exposures.

There has been a marked decrease in the size of all the tumors; that to which the ray was directly applied is almost normal in appearance, and in feeling. The opposite parotid swelling is still appreciable but much smaller. The remaining orbital tumor is not now visible, but can be found by careful palpation.

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DISCUSSION OF TUMOR SYMPOSIUM.

EDWARD B. HECKEL, Pittsburgh, Pa.: The practical point to be gotten out of a series of cases such as this, is the matter of diagnosis. One is rather struck with the simplicity of diagnosis after having listened to the readers, but as a matter of fact, orbital tumor is rather difficult to diagnose, and after the ordinary means have been exhausted, such as the examination of accessory sinuses by X-ray, etc., perhaps the most important point in diagnosis is palpation, because frequently we can palpate the mass and find out whether it is movable, and determine in what part of the orbit it may be. So the matter of diagnosis, after all, is difficult and requires some time for observation, to permit us to make up our mind as to just exactly where the mass is and its nature. Of course the real nature is not determined until it is examined microscopically.

The matter of the Kroenlein operation, or operations of that kind, may be determined by palpation. Many of these tumor masses may be removed without the Kroenlein operation, provided the tumor mass does not puncture the optic nerve.

The use of the X-ray and radium as postoperative treatment is very valuable. I recall a case I had the privilege of operating—a lymphosarcoma located in the upper, outer quadrant of the orbit. After removal, there was some swelling in the upper, outer angle of the orbit, which we treated by the X-ray. It disappeared about the third or fourth day. Some months after that, there was some swelling just below the eyeball. This was also treated with the X-ray and subsided very nicely. This

was some five or six years ago, and the patient does not show any symptoms whatever.

As regards the last paper, I suppose we have to assume the position of the agnostic, because the matter of Mikulicz' syndrome is in doubt, and the truth of the matter has not been determined.

DR. HAROLD GIFFORD, Omaha, Nebraska: The first paper treats of a form of tumor which I have never seen. The chief interest in the paper to me is the question of using the Kroenlein operation, and the technic employed in the operation. A number of years ago, I became convinced of the fact that the Kroenlein operation was not such an easy thing to do as the description would indicate. I found it was sometimes quite hard to get at this nice little triangle, and, in fact, in some cases impossible to do so. I also found that sometimes it did not go back into place as nicely as you would like it to do, so in one case I took out as much of the triangle as I could, and then cut the bone out and threw it away. The result was surprisingly good. There was none of the sinking in of the side of the temple that you would expect. I took out the entire outer rim of the orbit and quite a little of the outer wall, and there was practically no deformity, and the result of this led me to erect as an operation what I called definite resection of the orbital wall, in place of the Kroenlein in some cases.

In regard to Doctor Francis' operation, I have never seen this form of tumor, either. But this point must be remembered, that if you take out the lacrimal gland, it is important to stop the bleeding very thoroughly, because if blood is allowed to seep out into the spongy orbital tissue, you may have serious results.

Regarding the case reported by Doctor Benedict—the boy in whom an apparent sudden development of the tumor occurred—as I understood, he simply reported that the first sign of the tumor was the extreme protrusion which appeared suddenly overnight. Of course Doctor Benedict did not believe that the tumor had grown in that length of time, but such an occurrence might throw us a little off our guard as to the real nature of the case. With the presence of fever, it might suggest the breaking of an abscess into some part of the orbit. I recently was led to make a wrong diagnosis in the case of a man who had a blind right eye for a number of years. He came in with the history that while the eye had been blind for several years following destructive ulceration of the cornea, it had shown no signs of protrusion until five weeks before he came to me, when it suddenly began to protrude. He had marked exophthalmos and you could feel a very large tumor behind the eye, and in view of the history, I made a diagnosis of tumor of the antrum which had suddenly broken through the comparatively soft wall of the orbit and, pushing itself up, had pushed the eye forward. X-ray examination of the fundus and antrum gave no support to this, and operation showed a tumor, two inches in diameter, behind the ball and attached to its posterior wall. Section showed that the tumor had started in the choroid and, after producing only a thin layer of tumor within the globe, had burst through the posterior wall and grown to the back part of the orbit. It must have been growing there for a long time. The suddenness of the development of the exophthalmos was undoubtedly due, as in this

case mentioned by Dr. Benedict, to the eye suddenly escaping from the control of the orbicularis.

I have seen several blood cysts of the orbit, which were apparently due to leakage of one or more vessels back of the eye, with slowly developing tumor and without much impairment of the sight. These can be cured by cutting down on the tumor, investigating carefully with the finger, and then putting a thread through it. I find thread fixation to be the best form of fixation for an orbital tumor. You do not mutilate the tumor, and you can then proceed to dissect it loose with the aid of the finger.

In cases of other cysts of the orbit, such as dermoid cysts, which send prolongations back into the orbit, I believe these should be treated with trichloroacetic acid. I have cured four dermoid cysts in this way. In one of these, the tumor was the size of a pigeon's egg, at the inner angle of the orbit. That *might* have been dissected out quite readily, but in doing that I probably would have spoiled the superior oblique, so I cut into the tumor, swabbed it out thoroughly with trichloroacetic acid, closed it, and the case made a complete and permanent recovery. Unless you are certain that the tumor of the orbit is not a cyst, you ought really to put a thread through it and make a cut to see whether it is a cyst or not.

DR. W. R. MURRAY, Minneapolis, Minn.: The majority of tumor growths in the lacrimal gland can be classified as mixed tumors, and they have been considered as somewhat benign, or at least as possessing only a local malignancy, and not subject to recurrence after removal. That is true in a considerable proportion of cases. These gland tumors, however, do possess a malignancy, which may become active at any time. This is well illustrated in Doctor Francis' case.

Sometimes bilateral symmetric swelling occurs in the lacrimal glands, and when accompanied by a similar condition in the salivary glands, it seems to me constitutes the Mikulicz' syndrome. To my mind, the case reported by Doctor Scales is one of Mikulicz' disease. The prognosis in these cases is usually good, at least in the simple form. When there are complicating blood changes present, or a splenic involvement, the prognosis is correspondingly poor.

I believe Mikulicz has described his syndrome as bilateral, symmetric swelling of one or more of the salivary and lacrimal glands. Thursfield, writing of Mikulicz' syndrome, stated there were eight groups of cases in which this syndrome might appear, one of which is a congenital condition which is very rare; another, a Mikulicz disease, so-called, occurring in simple form, probably without any blood changes or any complicating involvement; another group attended by marked changes in the blood and lymphatics; also cases which were due to tuberculosis, syphilis and gout; and lastly, cases which were intermittent in character, with recurring periodic swelling of the glands.

The treatment of Mikulicz' disease, it seems to me, is nonsurgical. X-ray and radium have apparently given very good results.

The operative procedures described by Doctor Stieren and Doctor Benedict are very valuable, and in my experience at least, the Kroenlein operation is indicated in a comparatively small percentage of cases. It seems to me that the comparatively large number of cases of glioma reported

by Doctor Benedict confirm the statement of Verhoeff, that glioma is the commonest tumor of the optic nerve.

I agree with Doctor Gifford regarding the use of trichloroacetic acid.

DR. EDWARD JACKSON, Denver, Colorado: Two or three points emphasized in the papers are to me important enough to call attention to them again.

First, the diagnosis of tumor of the optic nerve. Dr. Stieren's case brought out the point that as to involvement of the nerve, or origin in the nerve, the symptom of impaired vision, or perhaps the date of impairment of vision—the period at which it occurred in connection with the other symptoms in the case—is most important. We can hardly have real involvement of the nerve without loss of vision, and usually complete loss of vision. We may have loss of vision perhaps from pressure on the optic nerve, but that will occur long after the exophthalmos. The early loss of vision, when exophthalmos is slight, or a history of loss of vision before exophthalmos, would pretty nearly fix an origin in the nerve itself.

The other point with reference to this case was, that the symptoms of choking of the disc and the changes in the vessels do not necessarily refer to what is going on inside of the nerve or its sheath. They may be due to pressure existing outside of the sheath, after the veins have left the optic nerve entirely. These two things—the changes visible with the ophthalmoscope and the loss of vision—are what we have to depend upon. As to actual involvement of the nerve trunk, the loss of vision must be the more important.

With reference to Mikulicz' disease, the paper of Doctor Scales and the paper of Doctor Lane that he referred to, bring the whole subject before us in the fullest manner—probably the best group of cases we have on the subject in the literature. Certain things seem to stand out. In the large group of cases reported, there are a number of different types. Among these, there is one in which the changes are confined to certain glands, principally the lacrimal and parotid glands, but to more than one gland. They do not pass readily into adjoining tissues.

The histologic changes described in Doctor Scales' paper, to my mind, place the condition pretty clearly as an infective granuloma. Because we know a good deal about syphilis and a good deal about tuberculosis does not negative the existence of other very closely allied infective granulomas, of which we do not know anything. In my opinion Mikulicz' disease is a disease *sui generis*, closely allied to tuberculosis, syphilis, and probably to cases of blastomycosis. The fact that this case improved, not in one particular gland but in all the glands, under potassium iodid, is suggestive. It might suggest blastomycosis. I have no doubt at all that the great mass of cases of blastomycosis dermatitis have been diagnosed as syphilis, and the ophthalmologist has been perfectly satisfied with his diagnosis. They get well under potassium iodid, but we know certainly they are not syphilitic. It is quite possible that there is another organism, closely allied, that gets well under potassium iodid, and to my mind, in the present state of our knowledge, that is the most rational explanation of these cases.

DR. C. NORMAN HOWARD, Warsaw, Indiana: I was especially interested in the papers dealing with tumors of the lacrimal gland, and wish to call attention to metastasis.

On December 15, 1914, I exenterated a carcinoma of the orbit, probably originating in the lacrimal gland. The patient died on July 16, 1922, having lived seven and a half years after the operation. The autopsy showed a slight return in the orbit, but an enormous amount of malignancy in the chest and abdomen. The preliminary report in this case was made before our Indiana Academy in 1919, and published in the *American Journal of Ophthalmology* in May, 1920. I am making this brief reference at this time, with the thought of submitting a more detailed report in the future.

The value lies in the fact that I believe there have been but seven other cases reported of lacrimal gland tumors followed by metastasis shown at autopsy. If I am wrong in this, I would be glad to be corrected.

DR. GEORGE F. SUKER, Chicago: In the question of tumor of the orbit, especially in the negro, the question of gumma must be eliminated. I have under observation, at the present time, a woman in whom the serologic test was positively negative, yet the eye was proptosed and immovable, and she was absolutely blind. She has improved very greatly under potassium iodid and mercury.

DR. WALTER R. PARKER, Detroit: I have had occasion to do the Kroenlein operation six times. On four of these occasions, if I had known all of the conditions present, I would have been forced to the conclusion that I could have done the operation desired without first having done an osteoplastic resection. In the other two cases, where the tumor was in the muscle cone, it seems to me it would have been impossible to do what I desired to do, without first having done a resection of the external wall.

DR. EDWARD STIEREN, Pittsburgh, Pa. (closing): The diagnosis of an orbital tumor is not hard—it is practically simple. The difficult feature is to decide where the tumor arises from, and by which route we shall remove it. I have removed orbital tumors and cysts by the direct frontal, by the so-called Knapp operation, and by Doctor Gifford's operation, which I think will answer in many cases. I look upon the Kroenlein as the operation of last resort.

In regard to Dr. Scales' case, some eighteen or twenty years ago I reported, in the *Bulletin of Johns Hopkins Hospital*, a case of bilateral enlargement of the lacrimal glands in a young negress. She had a general adenopathy and tubercular conjunctivitis. She made a complete recovery under proper dietary and hygienic management, without operation.

DR. WILLIAM L. BENEDICT, Rochester, Minn. (closing): I restricted my paper to tumors arising at the apex of the orbit. Primary optic nerve tumors unquestionably are very rare. Verhoeff states, that in twenty-seven years, they have had thirteen primary tumors of the optic nerve in the Massachusetts General Hospital.

It is impossible to get pathologists to agree as to the nature of one of the tumors—whether it is an endothelioma or sarcoma—and whether the presence of nerve fibers is necessary to confirm diagnosis. The question that presents itself to the surgeon is not so much what type of tumor he will find, but whether it can be removed and how.

The loss of vision that Doctor Jackson spoke of is important. In one case I showed the man had lost his vision eleven years previously,

but the proptosis apparently had not arisen until nine years afterwards. Doctor Gifford said the proptosis may come on overnight. In the case of the little girl that had the glioma, the proptosis was practically the same for the next six months after it first appeared and it came on overnight. As to whether it will disappear quickly, we are not certain.

The relation of blindness to the state of proptosis is sometimes misleading. Blindness may occur from tumors that arise anywhere in the orbit, and the proptosis may not come on until some time afterwards, although the tumor may have practically destroyed the nerve and gone back through the apex of the orbit.

The Kroenlein operation, particularly in children, is not difficult. It is only difficult in people with deep set eyes and very heavy malar and frontal prominences. Under the improved technic of Magitot, it is not a difficult thing to do, and where the intention is to save the globe, it is almost necessary to employ it. The frontal operation is, I think, frequently done in the wrong way. I have made the mistake—and I have seen it made—of going too low, and immediately being confronted with a lot of orbital fat. It is almost impossible to keep one's bearings under that condition. If the periosteum of the frontal bone or the malar bone is cut through and carefully elevated around the orbital rim, so that the contents are kept entirely within the orbital fascia, the incision can be made without losing control of the contents of the orbit. I think the Kroenlein operation is a distinct advantage, especially in children, and I think it is a mistake to do a frontal operation if there is much vision left. As to the cosmetic effect, 50 percent of people having orbital tumors will have ptosis following the operation, whether you do a Kroenlein operation or a direct frontal approach.

DR. JOHN L. SCALES, Shreveport, La. (closing): I would like to say that my thinking about Mikulicz' disease is punctuated with interrogation points. I am greatly puzzled, especially with reference to the question of direct infection in systemic disease. I can readily understand how direct infection might give an enlargement of one or more of the salivary glands, but just why it should happen that this infection should pick out both lacrimal glands and both parotids simultaneously and produce the result, is a thing I cannot understand. On the other hand, why, if it be a general systemic involvement, should you get such a pronounced involvement of these isolated glands as my case showed, and nothing else? I am afraid, with the present statistical information, I will never have another opportunity to study a case of Mikulicz, and I am wondering just why it should come about as it has.

With reference to syphilis, those who have to do with many negro patients always look for syphilis, when there is anything that might be reasonably ascribed to it. Old Doctor Cain used to tell us years ago—"Gentlemen, the negro race is divided into three classes: those who have had, those who now have, and those who will have syphilis." In this particular case, I made every effort to demonstrate syphilis, without any reasonably positive result, and there were no indications about the man. I have come to the conclusion, however, after years of observation, that the negro is very largely immune to syphilitic manifestations. I do not think that we find in them the pronounced results that we find in other races.

GRADUATED TENOTOMY OF THE INFERIOR OBLIQUE MUSCLE AS AN AID IN THE COR- RECTION OF CERTAIN FORMS OF SQUINT.

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Congenital palsy of the superior rectus, with secondary spasm of the opposite inferior oblique, in all probability is the type of motor anomaly which more frequently demands a tenotomy of the latter muscle for its correction than any other.

Duane reported a number of cases of palsy of the superior rectus before the British Medical Association in 1906, and in that paper formulated the operative indications for its relief. Later, Posey presented a paper on the same subject before the American Ophthalmological Society. Todd read a paper before this society several years ago. White has reported seventy-five cases, thirty-five of which have come to operation. Good results were attained in all but two. White has classified his cases as follows:

Class 1. Paresis of the superior rectus, with spasm of the opposite inferior oblique.

Class 2. Paresis of the superior rectus, with spasm of the opposite inferior oblique, and also spasm of the inferior rectus of the paretic eye.

Class 3. Paresis of the superior oblique, with spasm of the inferior oblique in the same eye.

I have studied eleven cases of spasm of the inferior oblique, of which number seven were secondary to a palsy of the opposite superior rectus. In the remaining four cases, I was unable to determine which muscle or muscles were primarily responsible for the spasm. Three of these cases were adults with convergent squint, in which contracture had undoubtedly taken place. In these three, a tenotomy of the inferior oblique aided materially in relieving the slight upward turning, which is the accompaniment of so many cases of convergent squint.

In locating the muscle or muscles responsible for the spasm of the inferior oblique, it is necessary to separate the cases into two classes: those in which diplopia can be elicited, and those in which it cannot. When the patient can recognize the

two images, the study of the field of binocular single vision will in most cases reveal the true nature of the anomaly. Duane's tangent plant cannot be improved upon for this purpose. I should like to emphasize one or two points in its use, which I have found helpful. The assistant who steadies the patient's head also holds the red glass, which I have fastened to a pointer about eighteen inches long. Holding the glass in this way makes it easier to keep the plane of it always perpendicular to the visual line as the gaze is shifted. I have also placed a black pin in the square which marks the limit of my own field in the six cardinal directions, for the purpose of having always a fixed point from which to measure the displacement of the images. It facilitates the making of the test and I believe increases its accuracy.

When diplopia cannot be recognized by the patient, a careful study of binocular rotations should be made objectively. While critical observations of the corneal images is essential to the proper study of the associated movements of the two eyes, I have found that it is sometimes easier to estimate the amount of lagging or upshoot, by observing the width of sclera showing between the lower limbus and lower lid margin when the eyes are elevated, and the estimated width of the cornea which has disappeared beneath the lid margin when they are depressed. This test should be applied routinely in the study of every case of squint of whatever form. The cover test applied in other positions than the primary one will frequently furnish valuable information. One of my cases of convergent squint, when tested with the head turned to the left, showed a deviation with the cover test of 50° , and when turned to the right but 40° , showing a greater weakness of the right lateral rotations than the left, and this, despite the fact that the tropometer showed equal temporal rotations, though subnormal. This difference was confirmed when the angle of the squint was measured on the perimeter.

The tropometer or perimeter when used to measure the monocular field of fixation in degrees gives helpful information. Quite as much stress, however, should be placed upon the facility with which the eye makes its rotations, as upon the actual number of degrees covered. Its weakness lies in the fact that it does not measure the verting power of any one muscle but of several, and for that reason it seems to be of more value in its application to the lateral than to the vertical recti and obliques. I have had the indicator on my instru-

ment fixed so as to permit of oblique rotations, but the readings are difficult to make, and I could never satisfy myself that they gave me much added information. Unless there is a margin of difference of 5° in the corresponding rotation of the two eyes, I do not place much reliance upon the findings. However, it is only fair to say that its value in diagnosis is greatly enhanced when used as a supplement to the study of the binocular rotations.

Finally, in diagnosing a spasm of the inferior oblique, it is well to bear in mind the possibility of a too high insertion of one of the interni. The late Dr. Todd, in a letter to the writer, brought out this point. I do not remember ever having seen it published. If the upshoot occurs when the eyes are rotated laterally and in the upper fields, and not in convergence, there is in all probability a spasm of an inferior oblique. On the other hand, if the upshoot persists when the eyes are in extreme convergence, a malinserted internal rectus should be suspected. Since we know that not all cases of squint are caused by an impaired or absent fusion faculty or ametropia, we are confronted with the problem of determining what muscle or muscles, by their faulty action, are disturbing the coordination of the two eyes. I am convinced, since studying some of my cases of squint more carefully, that I have greatly underestimated the role that paresis of the superior rectus plays in the etiology of certain types, and that a thorough search will frequently reveal the offending muscle.

If these cases are seen late—after contractures have taken place—it becomes increasingly difficult to make a correct diagnosis, and we are then confronted with the necessity of meeting conditions as best we may. In cases of either convergent or divergent squint, if there is a definite upshoot of one eye when both are rotated to the opposite side, I believe we are justified in tenotomizing the inferior oblique of that eye to aid the lateral adjustment, even though we cannot demonstrate to our entire satisfaction a paretic superior rectus or superior oblique, provided, of course, that the convergence test shows no malinserted internus.

Some operators prefer to advance the paretic superior rectus rather than tenotomize the inferior oblique. Theoretically, it is probably the better procedure, but from a practical standpoint I believe that it is very much easier to tenotomize an inferior oblique than to advance a superior rectus.

It seems to me, however, that the decision as to which operation we should perform rests upon other factors than ease of performance. If the individual upon whom we propose to operate can fuse, even though in a limited field, then I believe we should attempt to strengthen the weak muscle by an advancement rather than weaken the spastic one by a tenotomy. With the exception of a limited number of cases of divergent squint, the majority of the operable cases have never been able to fuse in any field, by reason of the peripheral obstacle to fusion which has existed. For this reason we are entirely justified in tenotomizing the inferior oblique, a much simpler procedure than an advancement of the superior rectus. Even in these cases it becomes necessary sometimes to advance or tuck the superior rectus, after having tenotomized the inferior oblique, in order to correct the vertical deviation in the primary position. While it is true that a tenotomy will abolish the upshoot of the eye, I have not been able to secure a vertical correction of more than 3° (arc) in the primary position. This is about what we might expect, if we accept Maddox' statement that the elevating action of the inferior oblique in the primary position is only 30% of the total.

There is one phase of the surgery of the inferior oblique which always has seemed to me to be a violation of a surgical principle when applied to an ocular muscle; namely, the complete severance of its tendinous origin in the superior maxilla. We know, when tenotomizing one of the recti muscles, that even though we completely sever its attachment to the globe, we can rely upon the check ligaments and the lateral expansion of Tenon's capsul to limit the effect of the tenotomy.

A tenotomy of the inferior oblique, however, calls for a severance of the tendon at its origin and not insertion, and so far as we know there are no fascial slips to hold the muscle after it has been released from its attachment to the bone. White reported one case of double origin in an oblique, but this must be regarded as an anomaly.

It seemed to me, that if we could lengthen the tendon at its origin by a series of cross cuts, such as has been suggested by Harman and later Todd for the recti tendons, we should be able to weaken the muscle sufficiently to accomplish our purpose without completely disabling it, allowing it to run wild and hoping that it would reattach itself somewhere.

On the last case which was operated upon, this was done. After the incision was made in the orbital septum, close to the bony rim of the orbit at the lower end of Terrien's line, the tendon was isolated on two small Steven's hooks, and three cross cuts made as close to the point of origin of the muscle as possible. Obviously, since the tendon is but 2 mm long, part of the cutting was done in muscle tissue. Of course, since there is no globe upon which these cut fibers can rest, and to which they become attached by scar tissue, as they do when a rectus tendon is lengthened, we must hope that sufficient scar tissue will be formed to support them and prevent them from tearing apart. However, even if they do this, we are in no worse plight than if we had deliberately performed a tenotomy in the first place.

Duane's article¹ describes fully the technic of the operation, and I shall not go over that ground except to emphasize one or two points. Whether the operation is performed under local or general anesthesia, complete hemostasis of the subcutaneous and orbital tissues adjacent to the operative field is very important. After the skin incision has been made and the muscle fibers separated down to the orbital rim, Meller's tear sac speculum will greatly facilitate the subsequent steps of the operation.

The opening in the orbital septum should be made carefully, as the tendon lies in close proximity to it and may be cut by a too free incision. In some cases, portions of orbital fat protrude, but while annoying this does not seriously interfere with the isolation of the tendon. Traction of the tendon rotates the eyeball up and out, and extorts the vertical meridian, illustrating beautifully the action of the inferior oblique muscle when acting by itself. An intracutaneous suture of fine black silk will reduce scarring to an almost negligible amount.

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DISCUSSION.

DR. H. W. WOODRUFF, Joliet, Illinois: Our attention has been called in this paper to an operation which, while not new, has probably been performed by comparatively few operators. The first suggestion of a tenotomy of the inferior oblique, which I have found, was by Landolt in 1885. He describes the operation of tenotomizing this muscle at its origin, the point of origin being exposed through an incision in the lower lid. Obviously, the effect of a complete tenotomy of an ocular muscle at its origin would be to completely paralyze it.

One who is interested in this subject cannot fail to be impressed with the report of these cases, the most notable of which is the one by Doctor Duane referred to by Doctor McCool—a case of torticollis relieved by tenotomy of the inferior oblique. Doctor Duane states:

"The most important of these indications are, first, paralysis of the superior rectus, congenital or traumatic, with fixation with the paretic eye and consequently secondary deviation of the inferior oblique in the opposite eye; second, paralysis of the superior oblique, with secondary deviation of the inferior oblique in the same eye. The former condition is rather frequent as a congenital anomaly. I think I have seen about forty cases. The picture is characteristic, so that the diagnosis can be made in some cases at the age of a few months. In a case of right sided paralysis, for example, as soon as the patient looks to the right, the left eye shoots obliquely upward. The patient avoids looking to the right, and to evade diplopia, either shuts one eye or turns the head to the right and tilts it. In looking to the right, and especially upward and to the right, characteristic diplopia develops."

Tenotomy of the muscle at the origin is quite different from tenotomy at its insertion. Tenotomies as usually understood are failures when used to correct malposition from paralysis of the opposing muscle. In many forms of paralysis, tendon transplantation or partial tendon transplantation may be considered. The ocular muscles lend themselves quite readily, and their action can be changed by a change in the point of insertion. After all, the most difficult point in many cases is to determine whether the case is one of paralysis, and what muscle or muscles are involved.

While I have never performed this operation, I shall not hesitate to do so when such a case presents.

We are indebted to Doctor McCool for calling our attention to this subject, and with better study of our cases and using the methods of investigation recommended by Doctor Duane, such cases will be discovered. I doubt, however, if in the phorias it will ever be indicated, and I can hardly see why the author has brought up the subject of partial tenotomy.

DR. WALTER B. LANCASTER, Boston, Mass.: With regard to the anatomy I should like to call attention to the fact that you run very little risk in fishing for the tendon of the inferior oblique. The muscle runs upward and backward; and under the muscle and above the floor of the orbit, there are no structures that you are likely to injure. The infraorbital nerve is protected in a canal. You can dip your hook down and scrape the surface of the bone and hook up the muscle without

risk. The lacrimal sac on the nasal side is about $1\frac{1}{2}$ mm. from the edge of the muscle at its origin. In a paper on the origin of the muscle, by Whitnall, which appeared last spring (*Anatomical Record*, May, 1921), he states that in 59 percent of cases the muscle comes within 2 mm. of the margin of the lacrimal sac, but if you dip your hook down on the temporal side underneath the muscle, you get the muscle up without much difficulty.

To remedy the trouble from orbital fat, we use a small spatula, about 10 mm. wide, with which the assistant pushes the orbital fat back and holds it back out of the way. Having raised the muscle on the hook, we put a stitch through it and tie, thus getting a secure hold. Next, cut the tendon off completely at its origin; then lift it up with this suture and make sure you have the whole thing, including all accessory fibers attaching the muscle to adjoining structures; then resect several millimeters from the end of the muscle, including the stitch.

In doing this operation, the most common failure is insufficient effect, and it has to be done over again, because some fibers of attachment were left. This device of putting a stitch in the muscle to pull it up and make sure of getting it all, was suggested by Doctor John Wheeler.

Nothing was said by the writer in regard to head tilting. I think that ought to be studied more fully. In my limited experience, tenotomy of the inferior oblique has not cured the head tilting as often as the reports in the literature would lead you to suppose. I rather suspect that the cause of failure is, that in cutting the inferior oblique and curing the upward deviation of that eye, you are attacking the eye which is not the fixing eye. Now, obviously, the fixing eye is the one which must dominate the compensatory tilting impulse. A nonfixing eye can no more make the patient tilt his head in compensation than a blindfolded eye, for vision of the nonfixing eye is suppressed; otherwise there would be diplopia. When there is head tilting in these cases, I believe it is due to the paresis of the superior rectus of the opposite eye. As Duane pointed out, when he announced the discovery of this form of squint, the primary defect is paresis of the superior rectus of the fixing eye, resulting in overinnervation of this superior rectus and consequently overinnervation of its associate in the opposite eye, which is the inferior oblique. If we wish to correct the head tilting, is it not probable that we must attack the exciting cause and try to help the weak superior rectus by an advancement operation? I look to see advancement of the superior rectus of the opposite eye take the place of tenotomy of the inferior oblique, when the effect on the head tilting is an important part of our purpose in operating. Tenotomy of the inferior oblique will have a place as a cure for the upward squint.

DR. WALTER R. PARKER, Detroit, Mich.: I have done this operation a few times, perhaps ten altogether. It has not been my experience that you can pick up the tendon and expose it with the ease the essayist would seem to indicate. Some times the muscle comes up easily, and some times one has a good deal of difficulty in differentiating it.

In regard to the relief from head tilting spoken of by Doctor Lancaster, I think the degree of head tilting is always relieved. It is not

always entirely cured, but the most striking thing in the result of this operation is the sense of relief the patient feels from being able to hold the head erect. Unfortunately, I have not obtained a perfect result in all cases. The relief from inshoot is very apparent, but the correction of the vertical error is not always so marked. I am speaking from an experience of ten cases, and of course that is a very small number on which to base an opinion.

DR. J. M. BANISTER, Omaha, Nebraska: The object of the tenotomy of the inferior oblique of the unaffected side is to relieve the diplopia, by causing an abnormal position of the image seen with that eye to correspond with the image observed with the affected eye due to paralysis of the contralateral superior rectus of this organ. Why cripple the normal movements of the healthy eye? Why not rather attack the paretic muscle of the affected eye?

In the essayist's paper, he mentioned the fact of the possibility of directing surgical attention to the paralyzed superior rectus instead of interfering with the inferior oblique on the other side. In many years of surgical work on the ocular muscles, I have been possessed of the idea that it would be far more rational to act on the positive side than on the negative.

Advancement of the paretic muscle should be the operation of election. The technical advancement has been mentioned as being too difficult to perform. The "tuck" operation with the buried catgut suture works most effectively in such cases, and I would earnestly recommend its use.

DR. JOSEPH L. MCCOOL, Portland, Ore., (closing): I tried to make it plain that the object of the partial tenotomy was to limit the effect in those cases in which there was absence of binocular single vision, and to reserve operation on the superior rectus to when binocular single vision was present.

The reason I spoke of care in going after the tendon in doing a tenotomy was not because there was any vital structures there, but I feel it is rather essential to make sure that you have the tendon. The last case but one upon which I operated, I cut down rather hurriedly on the septum orbitale, and before I knew it I had nicked the tendon. The thought came to me at that time, that if we were careful to dissect it out—have your hook on the tendon—make traction and get the action in the inferior oblique, it would be much safer.

It is not always such an easy thing to get the effect with a tuck in the superior rectus, more particularly if the superior rectus is paretic.

I did not mean to convey the impression that tenotomy of the inferior oblique is an easy operation, but that it is an easier operation than an advancement of the superior rectus. I never like to tenotomize the inferior rectus muscle, even though the effect may be guarded by a suture.

SOME PRACTICAL POINTS IN REFRACTION.

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Notwithstanding accumulated knowledge of theory and method, good refraction is by no means general. No proof of this statement will be asked by the older men, who are seeing much referred work. As at least 80% of our work is the measurement of ametropia, this statement stands as an indictment of our specialty. I think we may wisely consider some of the reasons for our failure to do better refraction and discuss remedies.

First among reasons, I would place the lack of adequate opportunity for thorough training in this country. We have a few, very few high grade postgraduate schools where good work is being done; we have more very poor schools, where after a few weeks men are given a certificate so stating, and who seem to believe, that they are qualified to practice ophthalmology and oto-laryngology. Another reason is undoubtedly a lack of interest in the less spectacular part of our work, and an impatience with what is regarded by some as the drudgery of ophthalmology.

Some men, either from lack of interest, lack of imagination or something else, seem to have no conception of what a good refraction means. They never become interested in the refinements of the work, and as soon as possible relegate it to an assistant, who has no greater interest and perhaps less ability. I have some friends, a large part of whose refraction is done by lay assistants.

For thirty-five years, my best energies and most of my working hours have been devoted to the study and correction of ametropia and the conversation of vision. I regard it as the most important work I do and love it. Naturally, I am seeing a good many patients who have not found relief, and my feeling is that most of the failures which I see are due to the lack of a rational method of procedure.

I do not presume to instruct those who have grown grey in ophthalmology. Please allow me to address myself to those younger men who may not as yet have acquired a habit of work. They will pardon me, I know, if some of the things to which I shall call attention seem very elementary and self

evident. I believe, however, that I can best explain what seems to me a "rational routine" by describing my own methods.

I do all my refraction by appointment, and insist upon adequate time to do it in my own way. I make the preliminary examination and take the history myself. In a carefully taken history and survey of the patient's general condition, will often be found the key to previous failure and valuable indications for special procedure. Some headaches, for example, may be due to eyestrain alone, but more are probably due to a combination of causes; a chronic blepharitis may be relieved by carefully selected lenses, but there may be in any case other conditions which must be regarded as contributory causes, and cleared up before relief is secured. If the patient who comes for glasses for the improvement of his vision has gained thirty pounds in a few months, and has lost his sexual power, one will be inclined to study his nerveheads carefully and take his fields. Such a history need not take long, and the questions will be suggested by the complaint of the patient; his appearance, age, etc. Only the essential facts brought out need be noted in one's record.

After taking the history, it is my custom to inspect and carefully note the appearance of the external eye and its appendages, the iris, the anterior chamber, pupillary reflexes, the excursions of the globes and the tension to the fingers. The vision is taken and the fundus examined through the small pupil. I never make a manifest refraction before using drops, and I always use drops at all ages. I have not the skill of some in the use of the ophthalmoscope, and have never been able to make a satisfactory examination of the lens, vitreous and eyegrounds through the small pupil. Even in suspected and known glaucoma, I have not hesitated to use cocain as a mydriatic, and have no cause to regret it. Of course, I always use eserine after the examination is over, and have the patient wait until the pupils have resumed their normal size. In elderly patients who have lost practically all of their accommodation, I use euphthalmin and cocain, 1% of each, and if these drugs do not dilate a rigid pupil, I use homatropin and cocain.

The prime essential in fitting glasses is a knowledge of the static refraction, which I do not believe we can determine without cycloplegia. I use atropin in the examination of all children, and of young adults who can give the time for it, especially if they have not found relief from glasses prescribed

under homatropin. I have frequently used atropin up to forty in such cases, particularly when prolonged rest seemed desirable, though I am seeing such cases less frequently than formerly.

For thirty years I have followed one plan in the use of atropin. I prescribe a 1% solution, and direct that two drops be instilled in each eye three times a day, until ordered discontinued. The patient is to lie down, or to have the head tilted well back, and the lower lid held away from the globe by pressure against the malar bone; one drop is put in one eye and the lid held firmly for twenty seconds; then a drop is put in the second eye, and the application repeated after two minutes. In very young or delicate children, only one drop is used in each eye. After the drops have been used one day, or three or four times, I make a careful fundus examination and use the ophthalmometer; my assistant does a retinoscopy, and after the patient has rested with the eyes closed for ten or fifteen minutes, I make the subjective test. The drops are continued, and the subjective test is repeated daily until the results are the same on two succeeding days. The advantages of the method are obvious. The patient gains from day to day in knowledge of what we want and how to help, his interest and observation are stimulated, until finally the results can not be changed. As a rule, three tests are made on succeeding days, occasionally four in a new or difficult case. In prescribing, I usually cut the sphere from 0.50 D. to 1.00 D. in hyperopia, except in cases of squint or high esophoria, when I give full correction. In myopia I always order the full correction, except in a few cases of very high myopia, when a patient sees just as well and more comfortably at the distance, with both eyes open, with less. The glasses are to be put on at once and worn constantly. Patients are always asked to report in a week or ten days, that I may say when the eyes may be used, and again in two or three weeks. If at this second visit the hyperopes are found to be overcorrected, the sphere is reduced until they get their best vision. Children are naturally more adaptable than adults, and the stage of adjustment is usually passed by the time the effect of the atropin is gone.

The question often arises: "How much of the time shall active boys and girls wear their glasses?" Of course, this depends largely upon the amount and nature of the defect, the symptoms complained of, the general health and the probable relation of the eyestrain to the general condition. Generally

speaking, glasses should not interfere with the normal activities of the child when out of doors, but should be worn constantly when in school and for all near work when at home, and also at the movies. In myopia we must be more strict, and I believe the glasses should be worn as much of the time as possible. Children should be provided with two pairs of glasses, in order that at least one pair will always be in perfect order, and they should report frequently to us or to the optician for adjustment. We all know that the only way to get the full effect of a lens is by looking through its optical center at right angles to its plane, and that our most careful work may be undone if the patient does not wear his cylinders as we have intended to have them placed. I am almost daily seeing patients who are in need of the most careful correction, whose attention has never been called to these facts, and whose glasses are in consequence seldom in perfect adjustment and do not give the relief looked for. Many of the newer frames made to hold circular lenses have no device to prevent turning of the lenses in the mounting. Such lenses should always be marked and examined from time to time to see that the axes remain where they should be.

Naturally, most of my patients are refracted under homatropin. After the preliminary examination, already described, the patient sits in a semidark room, and from three to six applications of a solution containing 2% of homatropin and 1% of cocain are made to each eye, care being taken that time is given for the cornea to become bathed in the solution before the lower lid is released. An hour and a quarter after the first application, I make the intraocular examination, and use the ophthalmometer. My assistant does the retinoscopy. The patient rests with his eyes closed until I am ready to make the subjective test. I believe the ophthalmometer is worth while and employ it as a routine. Carefully used, it gives us information too valuable to be ignored, and in some cases, where accurate retinoscopy is impossible because of cloudy cornea or lens, it is a very important help.

My consulting room is lighted from above by the indirect method. The shades are drawn during a refraction, and there are no disturbing side lights. The test cards are of black with white letters. They are hung on a black wall and illuminated perfectly and uniformly from the side. There is no direct reflex from the cards or the letters. I use a phoroptometer, which is carefully adjusted from time to time in order to secure perfect centering of the lenses, and the patient is urged

to make himself perfectly comfortable in the chair before the examination begins. Unless results are obtained very quickly, he is asked to rest his eyes frequently during the examination by closing them. I use an astigmatic chart, the old one with twelve spokes with three lines in each, and each spoke pointing to an hour of the clock. I would not be without it. If I do not get 6/5 quickly, especially if I have noted in the dark room that the refraction of the center of the cornea seems different from that of the cornea as a whole, I use the 3 mm. diaphragm. In refining my tests at the last, I have found Dr. Jackson's crossed cylinders of great help. If the patient is uncertain and inconstant in his answers, I immediately add a +3.00 D. lens to the correction so far determined; and test his accommodation as suggested by Dr. Duane; if more than 1.50 D. remains, I have more homatropin used, and have him wait half an hour and try again, or if it seems best I omit the usual application of eserine and have him come again the next morning for a second examination under homatropin. I use homatropin in adult patients at any age while much of any accommodation remains, and agree with Duane that the most important time is after 40. I seldom find it necessary after 55, and seldom use more than three drops in each eye after 48. I recall one patient, a physician, who at 66 was not made comfortable until I corrected under homatropin an astigmatism of +0.50 D., which was not manifest before. After completing the examination under homatropin or euphthalmin and cocain, I use eserine salicylate in 1/5% solution, making one, two or three applications at intervals of five minutes, and frequently having the patient wait until the pupils begin to go down. If the eserine causes distress, a hot towel usually gives relief. A drop of cocain will also help.

I am not unmindful of the possible danger of precipitating glaucoma, in patients predisposed, by the use of mydriatics. In an experience covering thirty-five years and many thousands of cases, I have had one case in which tension went up while the patient was in my office, and before eserine had been used. This was a patient on whom I would not now use homatropin: a Jewess, fifty years old, with rather shallow anterior chamber and beginning arteriosclerosis. Tension was easily controlled with eserine. I have seen her from time to time for several years, and she has not developed glaucoma. In another case, glaucoma developed after an examination under homatropin, but an acute influenza intervened. I had no opportunity to see the patient for twelve days, and I have never

felt sure that the mydriatic had anything to do with the glaucoma. Unfortunately, this patient became blind. As Duane says: "An eye which develops glaucoma under a cycloplegic is going to develop glaucoma anyhow. It is not an unmixed evil to have it develop under our eyes." I always make a postmydriatic test, after an examination under homatropin and cocain or euphthalmin and cocain and, using eserine as I do, I can make the second test after homatropin in two days. Probably the ideal thing would be to have the patient rest his eyes from all near work and come for the final test in four or five days, but this is not practical. Occasionally, it is necessary to cut a plus sphere prescribed in this way, but not often.

I make the postcycloplegic test by having the patient close his eyes, while I arrange before him the full correction as found under the drops. I then ask him to look at the letters and to read what he can. I then cover one eye and reduce the plus sphere or increase the minus one, until he gets his best vision and a perfect wheel, if that is possible. Occasionally, it is necessary to cut a plus cylinder or increase slightly a minus one, or even to change the axis of a cylinder, in order to secure the best vision of which the patient is capable. I know this is contrary to the practice of some good men, but it works, and I often think of a remark of De Wecker: "The best glass is not always the one which science dictates, but the one the patient accepts." After settling the matter of the distance glass, I test the muscle balance with the Maddox rod and note the result. Moderate errors are ignored for the time, unless the patient has not been relieved by a complete correction for his ametropia. I have seen as much as 4° of hyperphoria disappear in a short time with the use of a good correction, and have many patients with 8° or 10° of esophoria or exophoria, who do not know they have it. If hyperphoria or esophoria persist and are troublesome, I partially correct them by the use of prisms for constant wear. In exophoria due to convergence insufficiency, I often give relief with prism exercises. In divergence excess, I prescribe prisms or do a tenotomy, but I do very few tenotomies. After making the test with the Maddox rod, I test the accommodation—not in the exact way that Dr. Duane has used in his studies, but by the use of the sliding card of near types, on the rule of my optometer. I did not know until I adopted this plan, how many cases of subnormal accommodation I was overlooking, or in how many presbyopes the accommodation was not the same in both eyes. In ordering the reading correction in presbyopia

and subnormal accommodation, I strive to give my patient a range of 25 to 50 cm. (10 to 20 inches), unless he is a very tall man or one who prefers a more remote range. For cards and music, for bench work and some other things, a second correction is best, which will give a range of 50 to 75 cm. I always test the eyes separately, and often prescribe lenses which differ by 0.25 or 0.50 D. Dr. Duane's researches in accommodation have been of great practical value to me, but chiefly in impressing the fact that we must individualize. We can not prescribe glasses by rule of thumb. Each case must involve a study of the individual as well as the eyes. The general health, the sensitiveness and reaction to strain of all kinds, the amount and kind of eye work done, must always be taken into account. We must realize that an astigmatism of 0.25 D. may be a cause of great discomfort in some cases, while in others 1.00 D. may cause no symptoms. Any measurable defect should be corrected if it causes strain. It is my habit to ask all patients to report in a week, and again in a month after examination, in order that I may know the result of my work and make any readjustments necessary. My experience has convinced me that slight but real changes in refraction are occurring in many, if not most eyes, and I therefore advise all patients to come in once a year to be checked up. As a rule, I find sufficient change in two or three years to warrant re-examination, and in a few cases oftener. In closing this paper, I can not do better than to quote the ending of a paper on the same subject by Dr. Duane: "I think the whole matter of handling refraction cases may be summed up in these two maxims:

First, let us find out all we can about the eyes we are treating and the symptoms of which they are a part; using to this end every means that experience has proved to be helpful, never doing the work in a hurry, and remembering always that this work of all others requires patience, thoroughness, and accuracy.

Second, let us constantly bear in mind the fact that we are treating patients, not eyes; that we are handling human beings, not machines; and that we cannot do our best work unless in each case we put ourselves in touch with the individual man before us, showing sympathy for his troubles, consideration for his infirmities, and an understanding mind, to take in all the physical and mental factors which may affect his outlook on life, and determine his need for refractive or other correction."

A PLEA FOR MORE GENERAL USE OF THE CROSS CYLINDER.

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The correction of astigmatism enters into the majority of cases of refraction, and a careful refractionist spends by far the greater part of his time in estimating the strength and axis of astigmatic correction required.

Although the various astigmatic dials are as a rule fairly accurate for the estimation of the strength of astigmatic correction, once the axis is determined, there are unfortunately a goodly percentage of cases in which these dials are very unreliable. And in a very much greater percentage of cases, none of the commoner methods of estimating the astigmatic axis are sufficiently prompt or reliable for refined work.

There is, however, a simple piece of apparatus with which a close approximation to the strength of cylinder required can usually be obtained where the astigmatic dial fails; and with this same simple piece of apparatus, it is almost always possible to ascertain with great accuracy the axis required. I refer to the cross or crossed cylinder.

The cross* cylinder test for strength of astigmatism was first described by Dr. Jackson 35 years ago. The application of the same device for the determination of the axis was suggested by Dr. Jackson 15 years ago. Yet it is safe to say that relatively few refractionists are familiar with either method. This is especially true concerning the axis test, which in my opinion is much the more valuable and more uniformly reliable of the two.

For some reason the cross cylinder tests seem to have received insufficient publicity. By the majority of students, undergraduate or postgraduate, they are not readily appreciated without optical demonstration of some kind. In periodic literature, they have apparently been dealt with on three occasions only, twice in short papers by Dr. Jackson¹ and once in a paper by Dr. Schneideman². These three papers contain only one illustration.

*The word "cross" is here used in preference to "crossed," since the shorter word is more euphonious and carries the same meaning as the longer.

Those of us who through personal contact with Dr. Jackson or in other ways, have learned to use the cross cylinder tests freely find them all but indispensable. In my own undergraduate and postgraduate teaching, I devote more time to the cross cylinder than to any other detail of refractive technic. It is in the hope that I may be able to render these tests more widely available, that I have undertaken the writing of this paper and the preparation of a somewhat detailed series of illustrations.

Even where the cross cylinder tests are used, I sometimes find their technic to be incompletely understood; and in passing I may say that the brief reference to them contained in so excellent a work on refraction as that of Thorington is not entirely accurate.

The cross cylinder is a compound lens having a net minus strength in one principal meridian exactly equal to a net plus effect in the opposite principal meridian. It is equivalent to a lens on one side of which is ground a plus cylinder of a given strength, and on the opposite side of which is a minus cylinder of

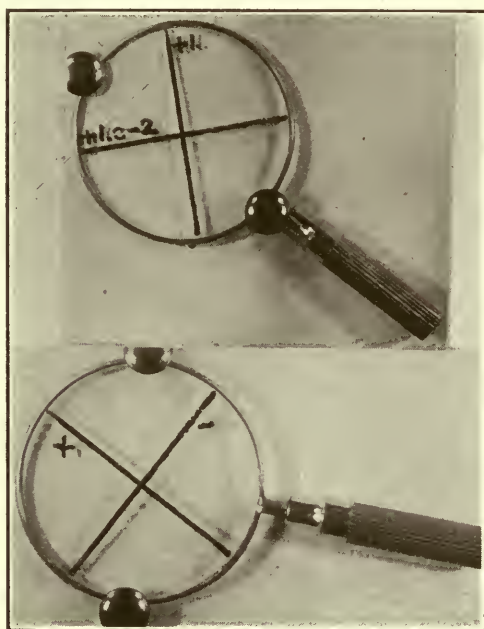


Fig. 1.

The upper photograph illustrates a cross cylinder made up of $+1$ D. sphere combined with -2 D. cylinder, and shows the correct mounting of the cross cylinder with its two axes at exactly 45° with the handle. In this and some other illustrations, the black lines are introduced to indicate the various details referred to in the legends. The lower photograph illustrates incorrect mounting of the cross cylinder, with its axes *not* at 45° with the handle.

the same strength, but at the opposite axis. In practice, it is usually ground as a minus sphere combined with a plus cylinder whose strength is twice that of the sphere. Thus we may have minus 0.12 sphere combined with plus 0.25 cylinder, minus 0.25 sphere combined with plus 0.50 cylinder; minus 0.50 sphere combined with plus 1.00 cylinder or minus 1.00 sphere combined with plus 2.00 cylinder. Of these the more commonly useful are the two middle strengths.

The cross cylinder should be placed in a circular mount, the angle of which must be at forty-five degrees with the two principal axes. Any deviation from this adjustment will render the manipulation of the apparatus inconvenient, and the test possibly inaccurate.

The cross cylinder is not placed in the trial frame, but is held in front of it, and the patient is given an instantaneous choice between two positions of the cross cylinder, the handle of which is rotated quickly between the examiner's thumb and index finger.

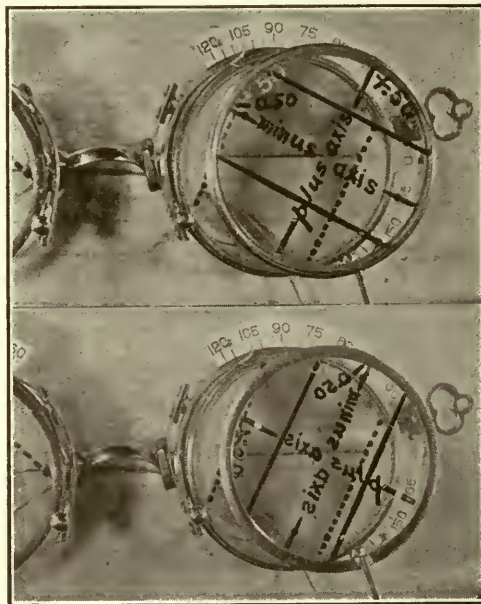


Fig. 2.

Illustrates the position of the cross cylinder before the trial frame in testing for strength of cylindrical correction. In each photograph, the cylinder in the trial frame is at axis 60°. In the upper photograph the plus axis of the cross cylinder is at 60°, whereas in the lower photograph the position of the cross cylinder axes has been reversed.

In testing for strength of astigmatic correction, the cross cylinder is held with first one and then the other of its principal axes coinciding with the axis of the cylinder in the trial frame, or, if there is no such lens in the trial frame, coinciding with any axis in regard to which the presence or absence of astigmatism is to be determined. The patient is instructed to look at the lowest line of letters which he is able to read even partially or with difficulty; and as the cross cylinder is rotated from one position to the other, he is required to say in which position he is able more readily to read any given line of letters. Or, as regards each position of the cross cylinder, he may be asked to say which is the lowest line of letters that he can even partly read. Either position of the cross cylinder may blur the type, and the patient's objections on this score may have to be put at rest.

Except in final tests, or in case of doubt as to his understanding of the test, the patient should not be called upon to



Fig. 3.

In this illustration the photographic camera has been made to imitate as closely as possible the appearances produced to the patient's eye during tests with the cross cylinder for strength of cylindrical correction. Myopic astigmatism axis 60° has been partly corrected with -0.50 cylinder axis 60° . (a) In front of the cylinder in the trial frame is held a cross cylinder (-0.50 sph. combined with $+1$ cyl.) with first its plus and then its minus axis at 60° . The fact that the second position makes the type more distinct indicates that the strength of the minus cylinder in the trial frame is to be increased. In (b) the strength of the cylinder in the trial frame has been increased to -0.75 D. The same test is made, and the greater distinctness with the second position of the cross cylinder again indicates an increase of the cylinder in the trial frame. In (c) the cylinder in the trial frame has been increased to -1 D. This time the cross cylinder renders the type equally indistinct in both positions, indicating that the strength of the cylinder in the trial frame is now correct.

name the individual letters aloud, since his doing so is apt to familiarize him too greatly with the test card.

The preferred position of the cross cylinder indicates the general character of the change which should be made in the cylinder in the trial frame. Thus, if the trial frame contains a minus cylinder with its axis at 60° , and the preferred position of the cross cylinder is that in which its minus axis is at 60° , the strength of the cylinder in the trial frame should be increased. The reverse would be true if the preferred position of the cross cylinder were that in which its plus axis was held to correspond with the axis of the minus cylinder in the trial frame. And vice versa, if the cylinder in the trial frame is a plus cylinder.

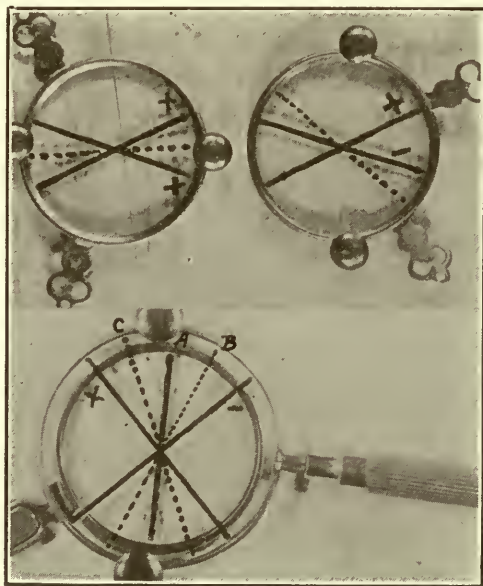


Fig. 4.

Illustrates the principle upon which the cross cylinder is used in testing for the axis. In the upper left hand photograph, two plus cylinders of equal strength are superimposed with their axes 45° apart. The resultant is a weak sphere combined with a stronger cylinder whose axis, as indicated by the dotted line, is midway between the two original axes. In the top right hand photograph, a plus cylinder and a minus cylinder of equal strength are superimposed, and in this photograph the dotted line illustrates the position of the minus axis of the spherocylindrical combination thus produced. The lower photograph illustrates what takes place when a cross cylinder is held with its axes at 45° with the axis (A) of a trial cylinder. If the trial cylinder is plus, a new plus axis is produced in the general position indicated by the dotted line C, whereas if the trial cylinder is minus, a new minus axis is produced in the general position indicated by the dotted line B.

Contrary to the statement contained in Thorington, the strength of cross cylinder employed, in conjunction with the particular line of letters which the patient is able to read with either position of the cross cylinder, does not necessarily indicate the exact increase or decrease required in the cylinder in the trial frame. An increase or decrease is indicated, but after each such change has been made, the test must be repeated to determine whether the increase or decrease has been sufficient, excessive or inadequate. The correction required is determined when the patient finds himself unable to read more letters with one position of the cross cylinder than with the other.

For "roughing out" the strength of cylinder required, this test is usually rapid and very fairly accurate, especially if the patient is called upon each time definitely to determine the very lowest line on which any letters are legible; although with all cross cylinder tests, the patient often needs to be carefully warned that he may never see so distinctly with the cross cylinder before the eye as he does without it. In the final fractions, and where the axis of astigmatism is nearly or quite vertical or horizontal, the test for strength is sometimes fallacious, in that the patient tends to prefer the position of the cross cylinder which produces a vertical rather than a horizontal distortion of the letters. For this reason, after I have found the approximate or final axis by means of the cross cylinder tests further to be described, I usually prefer to work out the final fractions of astigmatic strength by means of the revolving cross sold by one of our wholesale opticians under the name of the Camp Thomas astigmatic dial.

The cross cylinder test for axis is at first sight rather more complicated. It is based on the principle that two cylinders of like denomination, superimposed with their axes at an acute angle with one another, form a new cylinder of different strength, whose axis is somewhere intermediate between the two axes of the separate lenses.

If we lay a plus 1.00 D. cylinder upon another plus 1.00 D. cylinder, so that the two plus axes are 45° apart, the combination will be equivalent to a plus 0.25 D. sphere combined with a plus 1.50 cylinder, whose axis is exactly half way between the two separate axes. If the two cylinders are of unequal strength, the new axis will be nearer the axis of the stronger individual cylinder.

Suppose now that we have in the trial frame a minus 1.00 D. cylinder with its axis at 90° , and we wish to determine whether

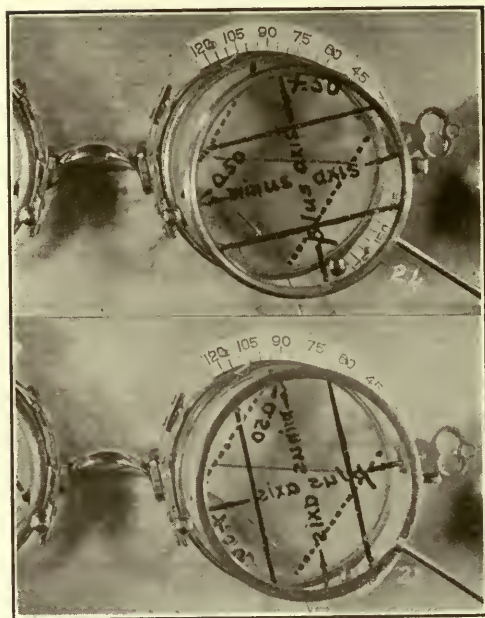


Fig. 5.

Illustrates the position of the cross cylinder in front of the trial frame in testing for axis. The axis of the cylinder in the trial frame, as indicated by the dotted lines, is here 60° . In the upper photograph the plus axis of the cross cylinder is at 105° , and the minus axis at 15° ; while in the lower photograph the relative positions of the axes of the cross cylinder have been reversed.

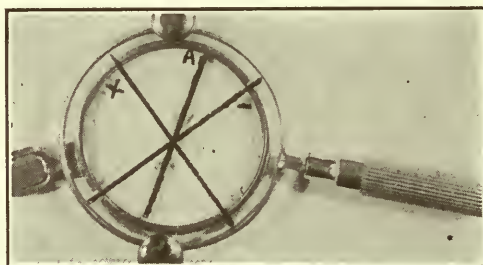


Fig. 6.

Illustrates inaccurate placing of the cross cylinder in testing for axis, the axes of the cross cylinder *not* being at 45° with the axis (A) of the test cylinder.

the axis should be changed in either direction from the exactly vertical position. A cross cylinder—say minus 0.50 sphere combined with plus 1.00 cylinder—is held with its two axes at 45° with the axis of the cylinder in the trial frame. If the minus axis of the cross cylinder is at 45° , there is produced before the patient's eye a new cylindric effect at an axis midway between 90° and 45° , that is at $67\frac{1}{2}^\circ$. If on the other hand the minus axis of the cross cylinder is at 135° , the resulting plus cylinder has its axis midway between 90° and 135° , or at $112\frac{1}{2}^\circ$. If the axis of the patient's astigmatic error lies nearer 45° than 135° , he may find the test type blurred by either position of the cross cylinder, but the blur will be less pronounced when the cross cylinder is held with its minus axis at 45° than when it is held with its minus axis at 135° . The change in position of the cross cylinder is again made by a simple rotation of the handle between the examiner's thumb and index finger.

The patient having expressed a preference as between the two positions of the cross cylinder, we must move the minus axis of the lens in the trial frame toward the preferred position of the minus axis of the cross cylinder. We have no exact indication as

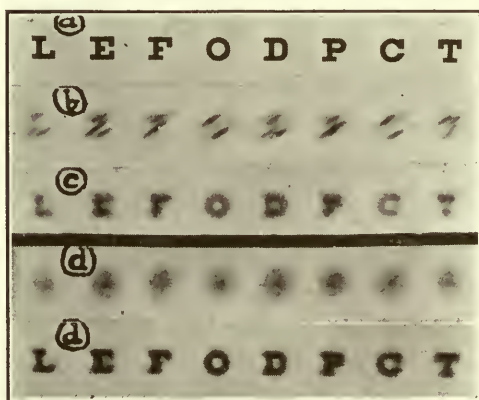


Fig. 7.

In this and the next figure the photographic camera has been made to imitate as closely as possible the appearances presented to the patient's eye during tests with the cross cylinder for the axis of the correcting cylinder. (a) is the appearance of the test type as photographed through the nonastigmatic photographic lens. (b) is the result of producing in the photographic lens a myopic astigmatism of 1 D. axis 60° . In (c) this oblique myopic astigmatism of 1 D. has been imperfectly corrected with -1 D. cylinder axis 90° . In (d) and (e) a cross cylinder (-0.50 sph. combined with $+1$ cyl.) is held with its minus axis first at 135° , and then at 45° . The vastly greater distinctness produced by the second position calls for a marked shifting of the minus axis of the correcting cylinder toward 45° .

to how far this change of axis should be carried. But the cylinder in the trial frame is moved arbitrarily any distance in the indicated direction, and the test with two positions of the cross cylinder is again made, only this time the axis of the cross cylinder must be at 45° with the new position and not with the original position of 90° .

At every new test, it is important that the cross cylinder shall follow the changed position of the cylinder which is in the trial frame. We may have shifted the position of the cylinder

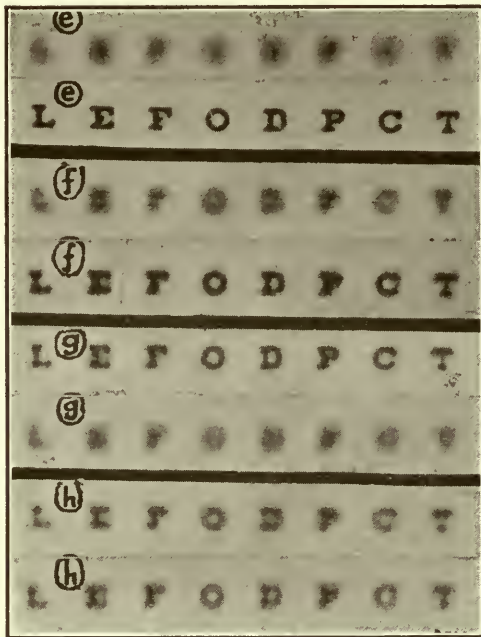


Fig. 8.

In (e) and (e) the axis of the minus cylinder in the trial frame has been moved to 75° . The cross cylinder is rotated with its minus axis first at 120° , and then at 30° . The greater distinctness produced by the second position calls for a further shifting of the axis of the test cylinder in the same direction as before. In (f) and (f) the axis of the minus cylinder in the trial frame has been moved to $67\frac{1}{2}^\circ$. The minus axis of the cross cylinder is first held at $112\frac{1}{2}^\circ$, and then at $22\frac{1}{2}^\circ$. Again, although less decidedly, a further shifting of the axis of the test cylinder in the same direction as before is called for by the greater distinctness produced by the second position. In (g) and (g) the axis of the minus cylinder in the trial frame has been moved to 55° . The minus axis of the cross cylinder is first held at 100° , and then at 10° . The greater distinctness produced by the first position indicates that the axis of the test cylinder has been moved too far. In (h) and (h) the axis of the minus cylinder in the trial frame has been moved back to 60° . The minus axis of the cross cylinder is held first at 105° , and then at 15° . The equal indistinctness of the type with these two positions of the cross cylinder indicates that the correct axis has been found.

in the trial frame either too far or not far enough. In either case the next test with the cross cylinder will tell us whether to go farther or to come part way back. But as we gradually diminish the range of alteration in the position of the cylinder in the trial frame, we shall at last reach a position in which the patient is unable to read any more or fewer of the letters with one position of the cross cylinder than with the other. We have then reached the desired point and have determined the correct axis required by the patient, subject to any change which may be obtained in checking up the strength of sphere and cylinder.

Like all other astigmatic tests, this one is more likely to be successful if the accommodation is relaxed, and, therefore, if whatever spherical lens is in the trial frame is so strong a plus or so weak a minus as barely to allow the patient to obtain his full visual acuity. Further, the patient must base his comparison of the two positions of the cross cylinder upon a study of the lowest line of letters which he can even partially or imperfectly read. It must also be remembered that the vision with the cross cylinder before the eye is very commonly less distinct than without it, and especially that at the final axis obtained, the cross cylinder blurs the vision equally in both positions of the test.

For eyes with good visual acuity, the test for axis may usually be made satisfactorily by means of the cross cylinder of minus 0.25 sphere and plus 0.50 cylinder. For the earlier stages of testing a high error, or sometimes as a check in unusually variable cases, the minus 0.50 sphere combined with plus 1.00 cylinder is useful. The minus 1.00 sphere combined with plus 2.00 cylinder is of value in relatively amblyopic cases.

In making the cross cylinder tests, the patient should usually not be asked whether he sees better with the cross cylinder or without it. What is needed is the choice between the two positions. Furthermore, there is almost never any advantage in checking the cross cylinder test for axis by means of the old-fashioned method of turning the cylinder in the trial frame in either direction until the patient decides that the vision is blurred.

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THE PRACTICAL SIDE OF THE OPHTHALMOMETER.

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This instrument cannot be included with the indispensable equipment, but this is true of a great deal of our paraphernalia that we would be unwilling to give up, and I am certain that some of its bad reputation is due either to faulty technic, or prejudice from experience with some of the early, defective models.

Aside from scientific considerations, the ophthalmometer can be used in a practical manner in most refractions, and if conveniently arranged, will be a time economizing device, instead of a scientific toy as is frequently charged. We are not expected to use all of our refracting room equipment in every case; however, in adopting a routine method for ordinary work, we select the instruments and methods that in our individual experience are the most efficient.

It is agreed that the patient should be disturbed as little as possible during the test, and should not be moved around the room, unless there is a definite reason for so doing. By using swinging brackets attached to convenient side walls and noiseless casters on smooth floors, all necessary equipment, including the ophthalmometer, for an ordinary refraction can be quickly and quietly placed before the patient, who is not moved from the original position. Under this plan, the actual time consumed in measuring and recording the corneal curvature of both eyes with the ophthalmometer averages about two minutes. Ignoring the scientific interest and value of the psychologic impression, it hardly seems possible to devote this much time to better advantage.

The actual findings have been found to be of little value in writing the prescription, but are useful in giving superficial ideas of what may be expected to be found later in the examination, and in checking the final result of other tests. If there are marked discrepancies, it is well to run over the test again, with a possibility of finding a clerical error in recording. Incidentally, I frequently find registered, especially in adults, a slight astigmatism with the rule, and if it is not

verified by the retinoscope and trial case, its presence is not understood and is ignored. Possibly this is due to a pressure of the lids, and is corrected by the ciliary muscle.

If the patient is approaching or is past middle age, and the ophthalmometric measurements show a plus astigmatism, axis vertical, and the trial case gives a plus cylinder, axis horizontal, I would suspect lenticular changes which would mean, of course, beginning cataract. In this connection, it has been noted by several observers, that in the preopacity stage of cataract, the refraction usually shows astigmatism against the rule, and advantage is taken of this idea in using the ophthalmometer in this suspected condition.

Information gained relative to the radii of curvature in hyperopia and myopia are of interest, but to me have not been of much practical value. The reflection of the images of the mires in a marked case of irregular astigmatism is startling, and in a mild case will show distortions and irregularities plainly, and will at times give a clue to this trouble. My experience agrees with that of Sheard's regarding ophthalmometric measurements in keratoconus, in that they are the most valuable test, and the instrument is of great service in following the course of the disease.

Leaving out the irrelevant features, I will refer to a few cases illustrating the value of the ophthalmometer as a time saver.

IRREGULAR ASTIGMATISM FOLLOWING OPERATION: Mrs. S. J. F., age 41. Left eye almost blind following removal of pterygium by another oculist four years ago. The ophthalmometer immediately revealed a very irregular astigmatism. Although the positions of the mires were so distorted that they could not be recorded, yet knowledge of the actual anatomic condition of the cornea justified satisfaction with obtaining a visual acuity of only 5/7.

IRREGULAR ASTIGMATISM DUE TO INDUSTRIAL VOCATION: Mr. J. H., age 55, miner. Vision in right eye poor for years, because of numerous minor industrial injuries. The left eye, the useful one, was enucleated two weeks ago, following a panophthalmitis from corneal ulcer. The right cornea was pitted with minute superficial grey spots, the scars of many battles incident to his occupation. However, all central media were clear and the fundus normal. The retinoscopic reflex was too indefinite to be of any value, and the trial case was uncertain, but the ophthalmometer at once gave a line or

the trouble, registering a rather irregular astigmatism of 5.5 diopters, plus, axis 90° . From this basis was easily worked out a correcting lens of plus-minus combination, which gave a useful vision of $1/2$.

KERATOCONUS: Mrs. J. A. B., age 25. Vision failing for six years, requiring frequent change of lenses. All refraction tests uncertain, especially of the right eye, the best vision of which being $5/20$. Because of the distorted images of the mires, a tentative diagnosis of conical cornea was made which subsequently proved correct.

KERATITIS PUNCTATA SUPERFICIALIS: Mrs. C. R. W., age 27. Suspicious teeth and tonsils. Some trouble with eyes since childhood, but worse for ten days and suffering acutely for two days. Eyes so sensitive that cocain was necessary for examination. Superficial structures inflamed, but no involvement of the iris, etc. The mires of the ophthalmometer well spaced and in alignment, but the individual mires are splotches resembling the cubist's art. Diagnosis confirmed with concentrated light and loupe.

Taking it for granted that the basis of this argument is logical, then also without reciting individual case records, the value of the ophthalmometer is apparent in the small pupils of senility, sensitive eyes, illiterates, amblyopes and unusual conditions.

THREE CASES OF ASTHENOPIA TREATED BY PSYCHOTHERAPY.

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Let me admit in the beginning that the title of this paper is open to criticism. The term *asthenopia* is used in its broad sense of weak eyes, inability to use the eyes continuously, without specifying the causative factors. The term is used for lack of a better one, and to avoid a lengthy explanatory title.

These three cases have this in common: in each there was a refractive error, which had caused some eyestrain and some local hyperemia and conjunctival irritation. These local symptoms were aggravated by use of the eyes, but were by no means serious in themselves. In each case, the patient attached tremendous importance to relatively trivial symptoms, not for the reason that the suffering was great, but because the patient feared it was a warning of disaster to come.

There is a considerable group of postoperative cases which belong in the same category as these here reported. It is of prime importance to see that the operated patient is in the right frame of mind during convalescence, lest he magnify the importance of his sensations and delay recovery by his fears. All successful surgeons, whatever their specialty, pay attention to this, though not always consciously.

CASE 1. An unmarried woman, 43 years old, complains that she cannot read even a minute or two without such discomfort that she has to stop. As her evenings are unemployed and she lives alone, the result is depressing and aggravates a tendency to morbid thoughts. During the day, she is actively employed making fancy lamp shades, at which she is so busy that she requires two assistants. She does both designing and making, and works eight hours a day at this fine, near work. She has had careful correction of refractive errors by competent ophthalmologists. Her general medical adviser is a well trained man, with special experience in neuropsychiatry.

After careful examination of the eyes, no new defect was brought to light. She had O. D. $-0.75 \text{ } \ominus \text{ } + 1.75 \text{ axis } 55^{\circ}$; V. = 6.18. O. S. $-0.75 \text{ } \ominus \text{ } + 1.50 \text{ axis } 130^{\circ}$; V. = 6/8. Muscle balance, less than $1/2^{\Delta}$ L. hyp., no es. or ex.; at the

reading distance, $3\frac{1}{2}^{\Delta}$ exo. Accom. = 4.50 each eye. Conv. good; pr. div. 5^{Δ} — 6^{Δ} . Overcomes 3^{Δ} pr. b. down, and 2^{Δ} pr. b. up, before left eye. She had been given glasses which varied from $-.75$ to 1 sph. with cyl. from $+1.50$ to $+1.75$ and with ax. 60° right eye, and 125° to 130° left. She had been given $+1.25$ sphere added for near work, but without benefit. Six months ago a tooth had been extracted, after which the head was better.

Fundi and media normal. Fields not taken. Lids, conjunctiva, and iris showed nothing to account for the trouble.

Since this patient was using her eyes on rather fine, near work for many hours a day without much inconvenience, and since careful examination showed no cause, it was obvious that the inability to read was not due to refractive errors, lack of accommodation, or muscle imbalance.

A little questioning, to draw out the patient's confidence, showed that she was dreading a serious breakdown of the eyes, which would compel her to give up her work and might mean permanent loss of ability to use the eyes. This was a serious handicap to her physician, who was unable to overcome her fears, since the oculists had not given her relief, and he could not but share her fear himself to some degree. The following considerations enable us to differentiate between hysteria and fear. There was no intolerable situation from which subconsciously she was finding a way of escape by inability to use her eyes, she was not trying to evade something which her sense of duty was urging her to do; that is, it was not a case of hysteria. Just the opposite was true. If the inability to use the eyes became general, and she had to give up her work, then indeed the situation would be serious. As she had already reached a point where she could no longer use her eyes for reading, it seemed to her not only possible, but probable, that before long she would be totally incapacitated, and she would far rather die. No wonder she was filled with fear.

After examining her with sufficient care and thoroughness to convince her that I was basing my opinion on adequate knowledge of the case, I assured her positively and emphatically that she had no disease of the eyes that would make her blind or lead to inability to use the eyes. I told her she need not give up her work, and that it was my belief that she would soon be able to read. In order to convince her more fully, I pointed out some of my reasons for my belief. I pointed out the fact that if she had a serious defect which made it impossible to use her eyes a

couple of minutes for reading, she could not possibly work on her lamp shades many hours a day. I explained that certain symptoms of discomfort which she felt when she began to read, which were thought by her to be signals which must be instantly heeded and would lead to disastrous results if neglected, were really nothing serious, and that it was her apprehension which incapacitated her. After explaining this at some length in as nontechnical and simple terms as I could, she suddenly interrupted me by saying: "You mean I have an anxiety neurosis." I said that was exactly what she had. She was pleased at the surprise she had given me by her technical knowledge, and explained that her physician had successfully treated her for a fear neurosis and had told her about it. (This concerned other functions, and had nothing to do with the vision.) This special knowledge and experience on her part made it easier for her to understand the nature of her eye trouble. It was not difficult to convince her that she had no serious eye disease, and was not threatened with blindness or incapacity as she had feared.

I saw her on April 12 and 14; and then wrote the following letter to her physician:

"I saw Miss — again today. There is no question about her eyes being sound. Their accommodation and other functions are perfect; the only trouble is, she cannot use them.

"I explained it to her, and she perceived at once that it was an anxiety neurosis. She became alarmed some time ago at some symptoms, and anxiety did the rest, although the symptoms were nothing serious. I think she grasps the idea, thanks to your training, and will cooperate fully.

"I told her to begin with about three minutes a day, and increase by one minute daily—paying no attention to the discomfort she would likely feel at first."

She was given a soothing collyrium to relieve the discomfort, to be used as often as she wished. The patient left the office very hopeful and enormously relieved of her apprehension; but was told to return in a week, since such cases are apt to have a reaction and again fall a prey to their old fears. Her progress has been entirely satisfactory.

If I had seen this patient without the benefit of the experience of the ophthalmologist who had studied the case carefully before sending her to me with his report, I should probably have given a guarded prognosis on account of the defective vision (only 6/8 with correction) plus the apparent incapacity for reading. If after adequate study and observation of the case, I had later decided that I could give a positive and emphatic favor-

able prognosis, it would have been far more difficult to convince her.

CASE 2. Man, 63 years old, Professor in a Western University, studying at Harvard during his sabbatical year. General health not good, due to some rather serious cardiac trouble. Referred to me by his oculist, because he could not read more than 15 or 20 minutes without such discomfort as to make him give up. Careful correction of refractive errors and suitable astringents for his slight chronic conjunctivitis, had failed to cure him.

Close questioning as to what made him stop reading showed that he felt a sensation of dryness and some scratchiness, and feared that if he disregarded these symptoms he would surely bring on serious eye disease, because these were the warning signs of eyestrain. It was his belief that if he had proper glasses, these symptoms would disappear, so that he could read without discomfort.

No opinion was expressed until I had completed a fairly thorough examination. This revealed no defects other than those already found by his previous oculists. He had distance O. D. -7.00 ; O. S. $-7.25 \text{ } \ominus -0.75$ axis 90; near, add $+ 2.50$.

It was possible therefore to assure him with great positiveness that there was nothing serious the matter, that the symptoms which he felt when he used his eyes were very common and could safely be ignored in his case. He was given my favorite soothing collyrium, and directed to use it many times a day. Whenever the eyes felt uncomfortable, instill a few drops. As time went on, he would find that he would not need to use it so often.

A slight increase in the power of the cylinder was accepted; and in order to aid the mental effect, one lens was changed. This is often of some importance, when the patient comes with a strong preconceived idea that a change in glasses is needed.

A week or two later he reported that he was laid up with a cardiac attack, and could not come to the office; but that he was using his eyes more freely than he had for many months. Subsequent progress favorable.

CASE 3. A man of 75, referred to me by his oculist for obstinate blepharospasm. He complains that he cannot read or use his eyes more than a few minutes after dark, because his right eye closes (blepharospasm). He has had to give up going out in the evening, as well as reading or other use of his eyes.

Here again careful search brought out no new defects. His H. and P. were well corrected; he had no muscular defects.

There was a moderate chronic conjunctivitis or conjunctival hyperemia. There was evidence of old iritis in the left eye, some lenticular opacities in both eyes. V.O.D. with $-1. \text{C} + 0.50$ axis 90 = 6/10+; V. O.S. = 6/40, not improved by glasses. He was thought by one oculist to have glaucoma, and used pilocarpin for a while. He has been using zinc sulphat 1:500, also syr. ac. hydriodic.

The significant feature was the ability to use the eyes during the day, and the occurrence of the blepharospasm only in the evening. I questioned him about his lighting arrangements; and advised certain improvements to make the lighting more like daylight in its diffuseness and freedom from harshness. His reading was restricted, and a soothing collyrium given to be used ten times a-day.

This gave no relief. Questioning brought out the statement that after reading a short time in the evening, he would feel a smarting or irritation of the conjunctiva, and then the eye would shut. Having once fallen into the habit of responding to the slight conjunctival sensation by a contraction of the orbicularis, the habit easily perpetuated itself and had become chronic. I thought there was a possibility that by combining confident assurance that he would recover with the use of a dilute local anesthetic in the eye, to remove the sensation which seemed to be the initial factor in the vicious process, we might get a start in breaking up the habit.

He was directed to instill a drop of the anesthetic (cocain 1:500) at 6:45 p. m., and then begin reading. In ten minutes, repeat the drop and read ten minutes longer; then repeat the drop, but stop reading. After three days increase the periods to 15 minutes. At the end of a week, call again.

At the next visit he was not inclined to admit much improvement, but his wife was sure he was better. He was told that he was on the right track, and would eventually recover, but that a trouble which had existed so long could not be expected to get well in a few days. After another week, he was told to begin to read without first putting in the drop, and use the drop only when the eye felt uncomfortable and inclined to close.

I did not see him again for over a year, when he came for something else, and I told him to go back to his former oculist. He had entirely recovered from his blepharospasm.

When the reaction to fear is through the subconscious mind seeking a way of escape from an intolerable situation, we get hysteric manifestations. These cases are not of that type. Nor are they like certain cases of malingering, in which the patient

greatly exaggerates some defect, in order to escape some situation, but does it consciously and intentionally.

In these cases which are here reported, the patient greatly exaggerates some minor pathologic condition, but exaggerates its significance or importance rather than its pain or discomfort. He fears that if his symptom is neglected, serious consequences will follow. He does it through ignorance and from good motives. His cooperation is usually easy to secure. If you can convince him that his symptoms are in essence trivial, that they are not warning signals which he neglects at his peril, you can effect rather rapid cure, especially if the manifestations are on the higher mental levels, as in the first two cases. If, however, the pernicious effect has involved lower levels, and established a bad habit or habits which have become quasi reflex, automatic responses, no longer requiring cooperation of the conscious mind, then, as in the third case, the cure is more uncertain and may take a long time.

Try to get a clear conception of just what has gone wrong, and why. Then you must use your ingenuity, with much patience and sympathy, to break up the morbid process. Do not make the mistake of telling the patient, or implying, that there is nothing the matter. Asthenopia or blepharospasm from fear are just as real as asthenopia or blepharospasm from uncorrected hypermetropia and astigmatism.

Successful treatment of this type of fear or anxiety state depends first on accurate diagnosis: you must be sure that you have not overlooked or neglected the treatment of any possible factor; second, you must convince the patient that you know what you are talking about, and you must secure his confidence and cooperation. The aid of consultants may be needed.

It is worth pointing out that it is much easier to handle successfully such a case, if, as in all three of these, it has been under the care of some other practitioner, who has carefully studied the patient and assures you that he has corrected all defects to the best of his ability, and does not see why the patient should not be able to use his eyes. You make a thorough examination and confirm his findings. You can then talk to the patient with more positiveness and evidence of your own conviction, than you might feel safe in showing if you were seeing the patient for the first time, and did not have other expert opinion behind you. If you make a guarded prognosis, and show caution and a disposition to hedge, the patient inevitably senses *your* fear and seizes upon it as a confirmation of his own—and the case is worse.

Again, if a patient has been watched and treated through some serious affection—say uveitis following cataract operation—it may be difficult for you to convince the patient that he can safely use his eyes and stop worrying. He knows how worried you were, and how you cautioned him not to do this or that. Now he feels some sensations which he thinks mean that he should still avoid the things you told him to avoid. If you arrive at the conviction that a case is of this kind, but only after you have studied and treated it for weeks or months, you are at a decided disadvantage when you attempt to convince the patient. He senses at once your change of attitude. He wonders whether your present opinion is any more likely to be correct than your previous one. If you find that you have not filled him with the necessary confidence and conviction, it is better to call a consultant to your aid. Obviously the doctors must agree among themselves.

DISCUSSION ON SYMPOSIUM ON REFRACTION.

DR. EDWARD JACKSON, Denver, Colorado: I believe a carefully evolved technic, persistent to the point of getting a real grasp of the case, is more important than any one method or routine procedure, even the application of cycloplegics. It is only the man who has an excellent technic who can use cycloplegics with success.

With reference to the cross cylinder, it is a method of analyzing the case, so that we can measure one meridian of the eye at a time. In using the test lenses, we find many factors that influence the acuteness of the vision. The cross cylinder gets rid of the spherical change that a change of lens might make, because it does not make any change in the spherical refraction. Used in a certain way, it tests only the need for change in the strength of the cylinder; used in another way, it tests only the direction of the cylinder that has already been tentatively selected and placed in the trial frame. It makes a change instantly—more quickly than a spherical lens held before the eye; much more quickly than a simple cylindrical lens placed before the eye and turned from one axis to the other. The cross cylinder carries a combination of cylinders from one extreme axis to the other, and by the immediate succession of the two extremes, makes the patient very much better able to compare them.

One point is, do not use too weak a cylinder. Use one strong enough, so that the patient will certainly recognize the change that is being made. That sometimes will require using one diopter change in the strength of the lens, when most patients would require only a one-fourth change in order to recognize the difference. Some patients must be almost knocked down by it, before they will fix their attention on the change. Until they do, their answers are only misleading.

With reference to the ophthalmometer, I agree heartily with Doctor Orendorff's statement that it is not an essential of our equipment, but it is of great value, in that it is a different way of making measurements. It differs from all our routine subjective methods; it is an independent

method. To be sure of our ground in these investigations, we should get the concurrent testimony of several methods, and the ophthalmometer is such an independent method, that it is of great value. The satisfaction of the patient will depend very largely upon accurate correction, and the patient must get accustomed to different conditions of vision. Until then he will complain of the glasses, so certainty with reference to correction is of great value.

It is also of great value after cataract extraction. Some of these cases are left with small pupils; many are left with different refractions in different parts of the pupil, so in those cases the shadow test is confusing. In the subjective method, with a large pupil different parts of which require different corrections, you must get the axis of the required cylinder objectively as accurately as possible; and as the astigmatism is usually high, the ophthalmometer gives the axis with great accuracy. This is of practical importance. I remember a case of a patient who had one cataract removed in Vienna by Mueller, and a cataract removed in Wiesbaden by Pagenstecher, and who came with a vision of not more than $3/5$ in each eye. When he had accurate correction—and the ophthalmometer assisted materially in this by giving a close approximation of the amount of astigmatism—he had a vision of $5/3$ in each eye.

Referring to psychotherapy. The symptoms of eyestrain are subjective, and complaints of subjective symptoms must be taken subject to revision as to their importance when further light is thrown on the case. In all such cases, whether having pain or disturbance of vision, the psychologic element must be considered. The mind of the patient is an element in all subjective phenomena, and must be met in the way the particular case requires. If there is one thing that is necessary to get good results, it is to raise the courage of the patient, but I am sorry to say that there are some members of the profession, who have been many years in practice, who have not learned that lesson. You would better take some risk with your reputation for exact prognosis, and give the most favorable prognosis that is at all probable than to discourage the patient and by the psychic influence interfere with the cure of the case.

As to the graduated exercises, illustrated in the last paper, for the relief of asthenopia that is not due to errors of refraction—the simple application of psychotherapy—it was the graduated exercises that demonstrated to the patient, as no argument will, that he can be what he thought he could not be.

DR. J. M. PATTON, Omaha, Nebraska: Doctor Westcott has outlined a program that, if carried out, would give the most satisfaction to ourselves and the greatest benefit to our patients. In his practice, he has his patients under control, he can see them as often as is necessary, and by so doing is able to eliminate certain things which may not be the best, but to which some of us have to resort. I refer particularly to the question of premydriatic tests. Dr. Westcott says he does not give these tests, but a great many of us see our patients only for a limited time, a day or perhaps two days at the most. They come from the surrounding territory and want to get away, so we have found the premydriatic test is of value, and in cases where we know we will see the patient only for one day, we give this test with great care.

I was glad to hear Doctor Westcott emphasize the use of eserine after the use of cycloplegics. As he has said, we do not often see a

case of glaucoma. In his wide experience, he has seen only one or two cases. But when these one or two cases can be avoided by the routine use of a method that is of value, they are just that many too many.

Dr. Orendorff's paper is very timely. Personally, I do not use the ophthalmometer in every case—perhaps I should. It is undoubtedly a great help in many cases.

My experience in the use of the cross cylinder has been largely limited to the ordinary trial case type, although I have also used the Rhodes double cylinder with satisfaction. Not understanding the cross cylinder as well as I should, I have felt that the disparity in the strength in the two axes was not an advantage. In other words, you reduce your sphere and increase the cylinder out of proportion to the correction in the trial frame. But I can see that this instrument would be of value in getting the axis in the manner described by Dr. Crisp. I think we who are not familiar with it should know more about it.

The final paper by Doctor Lancaster is perhaps the most important, and for this reason: We are too apt to think that we are oculists only, and forget that we are also physicians. Doctor Lancaster has emphasized to us, again, the fact that we cannot shift this responsibility. He not only emphasizes that these patients can be helped by the oculist, but he goes farther and shows how we may analyze these cases and give them relief. Personally, I very greatly appreciate these papers.

DR. E. C. ELLETT, Memphis, Tennessee: I will limit my discussion to one point—the use of simple cylinders in making the final test of the accuracy of a correction. All of our tests are good up to a certain point, and it is the last half or quarter of a diopter of error, and the question of five degrees or ten degrees in the axis of a cylinder, that taxes our skill and patience. The ophthalmoscope, the retinoscope, the ophthalmometer, and the subjective tests with lenses easily arrive at an approximate correction, and any means to arrive as exact results within these limits should be welcome.

The method I speak of is not new and is very simple, but many with whom I have spoken do not employ it. It does what many refractonists think they do with crossed cylinders.

If we take, for example, a correction in the trial frame of $+1.00 \text{ } \ominus$ with $+1.00 \text{ cyl.}$, axis 90° , by the use of the crossed cylinders ($+.25$ with $-.25$) we convert this into $+.75 \text{ } \ominus +1.50$, axis 90° , or into $+1.25 \text{ } \ominus +.50$, axis 90° , according to the position in which we hold the crossed cylinder. This is too great a contrast, and does not lead to arriving at an accurate result. If, however, we use a simple $.25 \text{ D}$ cylinder we can, by placing its axis parallel with the axis of the cylinder in the trial frame, in the simple example used, with a $+$ cylinder simultaneously increase the sphere and reduce the cylinder, and with a minus cylinder do just the opposite. Thus in a moment we can place the following combinations before the patient for selection:

$$\begin{aligned}
 &+1.0 \text{ D } \ominus +1.0 \text{ D axis } 90^\circ \\
 &\text{Add } +.25 \text{ axis } 90^\circ +1.00 \text{ } \ominus +1.25 \text{ axis } 90^\circ \\
 &\text{Add } +.25 \text{ axis } 180^\circ +1.25 \text{ } \ominus +.75 \text{ axis } 90^\circ \\
 &\text{Add } -.25 \text{ axis } 90^\circ +1.00 \text{ } \ominus +.75 \text{ axis } 90^\circ \\
 &\text{Add } -.25 \text{ axis } 180^\circ +.75 \text{ } \ominus +1.25 \text{ axis } 90^\circ
 \end{aligned}$$

The use of a .50 cylinder of course gives similar results. In the event of crossed cylinders being in the trial frame, one is increased or diminished without changing the other.

DR. DON M. CAMPBELL, Detroit: The only point I wish to make in connection with the use of the ophthalmometer is the satisfaction that I have had in the use of that instrument in cases of industrial injury, where I find reduction of vision that is not accounted for by a corresponding opacity of the cornea. It immediately raises the question whether the man is stating his vision correctly, and very often the use of the ophthalmometer will show in such cases a high degree of irregular astigmatism, due to the contraction of the cicatricial scar on the cornea. In that manner it has been of great benefit.

In the discussion of the question of subjective tests for refraction, it has often been mentioned that the patient is asked to read the lowest line he can see. The lowest line has a large number of letters, and the individual cannot concentrate his attention exactly upon all of these letters at one time. Therefore it has been found to be a great advantage, to have the individual pick out one letter in the lowest line he can read, and concentrate his attention on that. You have your trial frame loaded with what you consider the proper correction for this patient. There are only eight variations you can make from that correction as I understand it—two variations in your axis, two in your sphere, and four in your cylinder correction. The use of a sphere varying one-fourth to one-half diopter will soon decide the question of spherical correction; then use the cylinders as described by Doctor Ellett, and you will certainly get a most accurate estimate of the correct cylindrical correction. Maybe I do not understand the cross cylinder, but it seems to me that when you put the cross cylinder in front of a patient with this correction shown on the board, you do not change the refraction in one meridian, but in two. It seems to me an accurate application of the cylinders as described by Doctor Ellett gives us far more accurate and easy correction.

DR. H. V. WÜRDEMAN, Seattle, Washington: The reason the total amount of astigmatism does not correspond with the difference in the refraction of the major corneal meridians lies in the fact, that the cornea in all its curvatures is an irregular ellipsoid of three axes. Even in emmetropia the astigmatism of the optical zone, which is a comparatively small ellipsoidal area of 10° up and in and 15° in and down, varies in amount and direction of its axes from that of the visual axis. Then, too, the axes may not be at right angle. Outside of the optical zone, there are great and varying amounts of astigmatism. The eye does not look through the apex of the cornea; the corneal axis, the optic axis, the pupillary and the visual centers are in different places, and often separated as much as five degrees. Therefore, unless the measurements are made exactly or very nearly in line with the visual axis, there may be decided differences between the astigmatism found by the objective and the refractive examinations. An astonishing number of cases of conical cornea and irregular astigmatism will be found by routine use of the ophthalmometer.

DR. EDWARD J. BROWN, Minneapolis: The abstract in the March Ophthalmic Literature on Velonskiascopy by Trantas led me to experiment, and I devised what seems to me an improvement on Trantas' method.

Make a half millimeter perforation in a card, or use a small perforated stenopaic disc; pass this before the eye and look at a distant object, preferably a small point of light in a darkened room; if the eye is emmetropic, there will be no movement of the object, but if there be myopia or hyperopia, the light will appear to move with (myopia) or against (hyperopia) the movement of the perforation. The greater the error the more marked the movement.

The approximate axis and the amount of astigmatism is determined more accurately than by other methods. The Trantas test, or my modification, is of especial value in cases of corneas too dulled by disease to be practical subjects of skiascopy; also in cases where mydriatics are refused.

A chemist, 33 years of age, who had been invalided from a Government mustard gas factory because of irritable eyes, and who had been treated by several oculists in Kansas City, came to me wearing:

R. $+ .75$ cyl. axis 180° , vision 20/30

L. $+ .50$ cyl. axis 180° , vision 20/20

and able to do near work not more than an hour at a time. As he refused a mydriatic, he was refracted by the pinpoint perforation, which gave:

R. $- .75 = +1.50$ cyl. axis 180° , vision 20/15

L. $+ .50$ cyl. axis 180° , vision 20/20

DR. CHAS. A. BAHN, New Orleans, La.: I want to emphasize what Dr. Ellet has said concerning the necessity of studying the resultant combinations obtained by the use of cross cylinders. The cross cylinder, in most cases, changes the cylindrical equivalent twice as much as the spherical. The object of subjective testing is the determination of the one best vision lens by elimination of all other possible combinations. In most cases, the use of the cross cylinder in front of the trial frame contents produces a combination so far removed from the original that it practically needs no elimination. In plus on plus combinations, or minus on minus, greater than the test unit, the cylindrical and spherical components are inversely, but not equally, changed by the cross cylinder. In plus on minus combinations the effect is either to increase or to decrease both the spherical and cylindrical components.

The points mentioned are brought out in greater detail in "Subjective Eliminative Testing for Glasses," written by me and published in the Southern Medical Journal, 1921.

DR. SAMUEL G. HIGGINS, Milwaukee: When the supply of euphthalmin became exhausted at the Soldiers' Home, I resorted to the use of Burroughs, Wellcome & Company's homatropin and cocain discs. One disc, containing $1/50$ grain of homatropin and cocain, is placed in the eye, routinely, before I make a study of the lens or fundus. It has been used on some 300 men, varying in age from under 30 to over 80 or 90 years. In no case has this amount of homatropin caused any rise of tension or glaucoma or other complications, and I have a better mydriatic effect than I formerly obtained with euphthalmin or cocain. I am inclined to continue the use of homatropin and cocain discs.

DR. JOSEPH L. MCCOOL, Portland, Oregon: The meat of Doctor Crisp's paper seems to me to be the application of the cross cylinder in finding the axis of the astigmatism. Many men use it to determine

strength, but few for axis. I have used it for both for fifteen years, and can recommend it both for rapidity and accuracy in finding the axis, and what is far more important, the ease with which patients can detect the slightest shade of difference.

DR. GEORGE F. KEIPER, Lafayette, Indiana: The ophthalmologists owe a very great debt of gratitude to Doctor Edward Jackson for the introduction of the cross cylinder into refraction work, although it seems that very few use it, either because they do not know about it, or do not know how to use it. Personally, I have used it ever since I entered practice, with the greatest satisfaction.

The cross cylinder rests upon this principle: That every cross cylinder can be resolved into a corresponding sphere and cylinder. The cylinder is the sum of the two with the sign and axis of one, and the sphere is the other cylinder with its sign. Thus you may have two possible combinations to work from. You add to the cylinder but subtract from the sphere in the trial frame, or vice versa. If that rule is observed carefully, no trouble will ensue when this little convenience is used.

Doctor Orendorffs' paper on the ophthalmometer is timely. No matter what aspersions have been cast on this instrument, it is at times a very great time saver, besides giving us an independent means for reading off the astigmatism of the cornea.

DR. VERNON A. CHAPMAN, Milwaukee: Those of us who are using the newer phorometers must be careful in testing out the phorias to see that the patient is not touching that eye cup with the eye. Some of the newer phorometers do not have a headrest; they have an eye cup. The slightest pressure on the upper or lower part of the globe produces a vertical phoria. I have seen quite a good many cases of patients wearing prisms base up or down since the newer phorometers have come out, patients in whom no hyperphoria could be found by careful tests. If you let them take their own position they will throw the cup against the upper or lower part of the globe, immediately producing a vertical phoria. One must be careful to keep the patient back from the apparatus far enough so that the eye cups do not touch the globe coverings, or pull upon the lids, or displace the skin about the orbit.

DR. HAROLD BAILEY, Springfield, Missouri: Doctor Westcott spoke of the advantage of having a comfortable chair for the patient during a refraction. This is true as a rule; however, all of us have cases occasionally, in the refraction of which such a chair might be improved were a couple of tacks added to the seat and a sharp spike inserted in the headrest. Such cases require some added stimulus to keep them awake.

The mental state of a patient who has been kept waiting in your reception room for several hours is often not conducive to good refraction. It is a mistake to try to refract a patient when he is tired. It is equally true that the tired surgeon is a poor refractionist. To obtain the greatest measure of success, one should be just as alert in doing a refraction as in a cataract operation. It is a good plan to let your patient lie down and rest while the homatropin is being instilled. He will feel refreshed for the work that is before him, and the harder the work, the better his glasses will fit him.

These small points help us toward that perfect end result we are so anxious to obtain.

REPORT OF TWO CASES OF SYMPATHETIC OPHTHALMIA PRESENTING UNUSUAL FEATURES.

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As a preface to the report of the two cases whose histories I wish to relate, it may be permissible to recall a few of the established facts in regard to sympathetic ophthalmia. If this name does not suit those who seek to find one more descriptive of the condition, it might be said that it is more fitting than the terms "cataract" or "glaucoma" or some other common names of common affections, and its meaning is at least well understood.

By sympathetic ophthalmia we mean an iridocyclitis in one eye following a similar affection of the other eye. The exciting eye usually owes its affection to traumatism. The symptoms in the exciting eye, or sympathogenic eye, are very variable, though the pathologic changes, which will be again referred to, are more constant, and these symptoms may appear at almost any time, from a day or two to many years after the trouble originates in the exciting eye. It is not necessary to go more into detail on this point, but cases have been reported arising at almost any time after the primary injury was received. Sympathetic ophthalmia is usually thought to be prevented by removal of the sympathogenic eye, but its removal after sympathetic trouble starts in the second eye is not always followed by good results.

Because it has an important bearing on the cases to be reported, the symptoms of the onset of sympathetic ophthalmia will be quoted from Gifford's splendid article in the *American Encyclopedia of Ophthalmology* (p. 12369): "The first symptom is dimness of sight. This comes on insiduously, without pain or premonitory symptoms. Within 24 hours, however, there is generally added a very slight circumcorneal congestion, with a few fine deposits on Descemet, and very slight iritic adhesions. The adhesions become firmer, deposits on Descemet increase, with complete occlusion and seclusion of the small pupil, and iris bombé. If the pupil has been partially dilated, peripheral adhesions form later, binding

nearly the entire posterior surface of the iris to the lens." Fuchs has the following positive statement (Text-Book p. 142): "In conjunction with an insignificant reddening of the eye, deposits—which are never wanting in the beginning of a sympathetic ophthalmia—appear upon the cornea."

CASE 1. Miss B., a school teacher, age 28, was seen Dec. 29th, 1921, six hours after an automobile accident, in which the left eye was cut by a piece of glass. The wound passed through the cornea from 2 to 7 o'clock, wounding the iris and lens, and extending about 2 mm. into the sclera at each extremity of the wound. The ball was collapsed. As the wound was clean cut and not infected, and the ciliary body was not certainly injured, it was thought advisable to try to save the eye. The conjunctiva was dissected from the ball all around the cornea and brought together in a horizontal line, so as to completely cover the cornea. The ball regained its normal fullness, and the stitches were left in four days. On the eighth day, the conjunctiva had slipped back to its normal position, except at each extremity of the wound, where it was attached a little way onto the cornea. The cornea was clear and the anterior chamber reformed. The patient was sent home, and returned on Jan. 13th, fifteen days after the accident. The right eye was normal, vision 20/20 and J. 1. The left eye had only light perception and was soft; the scar was depressed, but there was no pain. No reflex was obtainable. Enucleation was advised for prophylactic and cosmetic reasons, and was done on Jan. 14th, sixteen days after the accident. An 18 mm. glass ball was implanted in Tenon's capsule. The muscles were not sutured, but a buried purse string catgut suture was placed in the cut edges of the capsule, and the conjunctiva closed separately with silk. On Jan. 17th, the conjunctival stitches were removed, and on the 21st, one week after the operation, the patient again went home. On Feb. 1st, 33 days after the accident and 18 days after the enucleation, she returned with the statement that the right eye had been uncomfortable for 3 days, and was a little red to the outer side. The ball was a little red, pupil active and dilated fully to atropin. All the media were clear, no deposits on Descemet, fundus normal. Vision 15/70, with $-1.25 \text{ C} - .50 \times 180 = 20/20$. Feb. 2nd., vision 15/200, $-3.00 \text{ C} - .50 \times 180 = 20/20$. Cornea, iris and fundus normal. Faint flush of eye to outer side. In consultation, Dr. J. L. Minor thought there was a beginning iritis, but no sign of sym-

pathetic ophthalmia, and while he did not see what harm the glass ball could do, he suggested its removal. This was done that afternoon. It was well covered with firm tissue and was a satisfactory implantation operation, with no signs of infection or undue reaction. The stump had not been painful or tender. Feb. 5th, vision 12/40, not improved by glasses. The retina assumed a whitish edematous look, with full veins. Feb. 6th, V. = 8/200, retinal edema more marked. Feb. 7th, some streaks of exudate appeared on the anterior capsule, and some vitreous opacities were noticeable. The retina was seen to be detached peripherally all around. Feb. 20th, vision perception of light. Impacted feces. 2/21, catheterized. Eye a little red and painful. Few hemorrhages on the iris, and translucent exudate in the outer two-thirds of the anterior chamber. 2/24, exudate smaller, 3x1 mm. and dense. Vision clearer. Retinal detachment less. March 4th, eye white. Pupil dilated (7 mm.). A little exudate on capsule. Anterior chamber deep. Tension a little reduced. June 2nd, vision 3/200; by looking down, 6/200. The eye is full and white. Pupil 8 mm. and filled with a thin white membrane. Good reflex all over fundus, and now and then a vessel can be seen. Retina reattached. A mass of pigment is seen to nasal side of disc, and a few faint deposits on Descemet. Anterior chamber of normal depth. Tension too low for the tonometer to measure. The field was normal in extent, but showed a relative central scotoma, about 15° in diameter.

The general physical and laboratory examinations showed the following: Urine; specific gravity 1006, negative for albumin, sugar and casts; few pus and epithelial cells. Blood; 17,000 whites, Wassermann negative. Tonsils; suspicious. Enucleated under local anesthesia, Feb. 14th. Nose and sinuses negative. X-ray of teeth negative.

The treatment was atropin, dionin, hot applications, large doses of salicylates, mercurial inunctions, pilocarpin sweats and thyroid extract. The salicylates were given in the dose of one grain per pound of body weight in 24 hours.

SUMMARY. A perforating wound of the left eye passing through the cornea, wounding the iris and lens and possibly the ciliary body. Conservative treatment for two weeks, and then enucleation for prophylactic reasons, with implantation of a glass ball in Tenon's capsule. Eighteen days later, failure of vision in the fellow eye, with development of myopia, then uveitis and retinal detachment. Recovery in four weeks

with greatly impaired vision and a soft eye, but a reattached retina.

Treatment.—Removal of glass ball, atropin, salicylates, mercury etc.

Examination of the enucleated eye showed the wound healed. It extended 4 mm. into the sclera above, and a shorter distance below. The pathologic report, from the Section of Ophthalmic Pathology of the Army Medical Museum, is as follows:



Fig. 1.

Fig. 1. Case 1. Section of whole ball showing the corneal wound, partial absorption of lens and flat separation of the choroid. (x. 7.)

HISTOLOGIC EXAMINATION.

Sclera: Some lymphoid infiltration around episcleral vessels.

Cornea: Thickened, wrinkled; perforation near center, protruding through it prolapsed, granulating iris; adhesion between iris and cornea only in posterior half; in anterior half, groove between it and cornea; nasal edge of corneal wound, only slight reaction and proliferation; on temporal side, active scar and kerotoblast formation; leucoma; Descemet's membrane on temporal side imbedded in granuloma; endothelium intact.

Anterior Chamber: Homogeneous coagulum in temporal half without cells; the angle is free; some infiltration of pectinate ligament and around Schlemm's canal.

Iris: Small posterior synechia of temporal pupillary margin to lens remnant; some diffuse increase of nuclei, but only one minute focus of lymphoid cells, around a vessel in temporal part; the sphincter on nasal side is absent, excised (?); nasal part of iris adherent to granuloma.



Fig. 2. Case 1. Perforating wound of the cornea and lens with anterior synechia. Extreme edema of the cornea. Corneal wound closed by organized tissue, wound in lens capsule closed by proliferation of capsular epithelium, and lens matter slightly infiltrated with pus cells. Anterior part of vitreous infiltrated with serum and numerous large round cells, many of which are pigmented. Ciliary body densely infiltrated with round cells. (x. 10.)

Lens: Small, shrunken, nearer to temporal side; shows double perforation of capsule; cataract; beginning capsular cataract; around capsule, near perforation and inside of it, some pigment cells, lymphoids, and many leucocytes.

Ciliary Body and Choroid: Greatly detached from sclerotic by homogeneous coagulum, which also invades choroid; about 2 d.d. to nasal side of disc, focus of lymphoid cells in outer layer; on temporal side three or four minute foci in outer layers, apparently of plasma cells.

Retina: Slight detachment; vacuolization of sensory epithelium; mantle of lymphoid cells around all vessels.

Optic Nerve: Decided swelling of disc, retina on each side being pushed away from scleral opening, more on nasal side; superficial layer of disc, infiltration around central vessels with lymphoids, leucocytes and eosinophiles; the lamina cribrosa in normal position, behind it also some increase of nuclei, but cannot be identified because of evidence of traumatism. (About one d.d. to the temporal side of disc



Fig. 3. Case 1. Edema of optic disc and surrounding retina, and marked perivascular infiltration of the disc with lymphocytes. (x. 15.)

on external surface of sclera, cross section of ciliary nerve with a ganglion cell.)

Vitreous: Detached and crowded behind lens, containing red corpuscles, lymphoids, leucocytes and shadow cells.

Diagnosis: Perforation of cornea, beginning adherent leucoma, posterior synechia, traumatic cataract, capsular cataract, slight reaction in iris and choroid; optic neuritis.

In connection with this report and the one to be read of the specimen from the second case, the following account of the pathology of the sympathogenic eye is quoted from Gifford (loc. cit.).

"In the vast majority of sympathogenic eyes, the greater part of the uveal tract is found to be the seat of an infiltration with small mononuclear cells (lymphocytes), more or less nodal in character, the grouping not infrequently occurring around the blood vessels. In the later stages, these nodes become fused into general lymphocytic thickening (often so great that it can be seen in sections with the naked eye), with groups of epithelioid and sometimes giant cells. In the stage at which such eyes are commonly examined, the infiltration of the iris and ciliary body is apt to be somewhat



Fig. 4. Case 1. Slight lymphocytic infiltration of the choroid, chiefly around the veins. (x. 12.)

diffuse, while in the choroid the nodular character of the inflammation is more pronounced."

There is no question in my mind, nor in the mind of several other Fellows of the Academy who saw this patient, that this patient had sympathetic ophthalmia. The first sign was the failure of vision, relieved by minus glasses, as is usual in iritis. The increase of the myopia from 1 D. to 3 D. in 24 hours evidences the rapid increase in the density (increase of refractive index) of the media. The peculiar features of this case are:

1. The development of sympathetic ophthalmia two weeks after a prophylactic enucleation, and one month after the injury.

2. The development of a typical iritis spongiosa during the course of the disease.

3. The absence of deposits on Descemet's membrane during the height of the attack, though they were present on the anterior surface of the lens.

4. The retinal detachment, which came on in the course of the disease and disappeared. Coincidentally the anterior chamber, which had been abnormally deep in its periphery, became of normal depth.

The question of the influence of the glass ball in Tenon's capsule on the occurrence of the sympathetic trouble in the other eye must be considered. If the operation had been a Mules' operation, the possibility of retention of a certain amount of uveal tissue and its harmful effects would have to be taken into account, but in this case the eye was removed with a satisfactory amount of the optic nerve, and the ball was implanted in the capsule of Tenon. At the time of the removal of the ball, the orbit was not painful or inflamed or tender, and the removal demonstrated the fact that the tissues were well healed over the ball, and the surgical conditions were in every way perfectly satisfactory. It is my opinion that the presence of the glass ball had nothing at all to do with the occurrence of the sympathetic ophthalmia.

1. *The development of sympathetic ophthalmia after the removal of the exciting eye.* While the literature contains numerous references to cases of sympathetic ophthalmia developing after enucleation, it is likely that many are, as I was, inclined to think the danger of this complication as safely passed when the eye is removed before the appearance of any trouble in its fellow. It takes a personal experience to impress these things on one. As Gifford expresses it (American Encyclopedia of Ophthalmology p. 12392) "the security afforded by the various preventive operations is only relative." That danger exists after evisceration and after Mules' operation, or after opticociliary neurotomy is not surprising, since more or less of the offending eyeball is left, but certainly one has done all he can do when an enucleation is performed, as already stated. I am not inclined to blame the glass ball implanted in Tenon's capsule with any part in the production of the sympathetic ophthalmia in this case. In no way, in

the light of what we now believe as to the role of uveal tissue in the cause of sympathetic ophthalmia, could this implant have had anything to do with it. Nevertheless I felt that it should be removed.

A varying number of cases has been collected of sympathetic ophthalmia occurring after enucleation, some 70 or 80 in fact. It is hard to be accurate in the count, as cases are duplicated in collecting and reporting them, and some are so poorly reported that we can not tell just when the sympathetic ophthalmia developed. The time of appearance of the sympathetic ophthalmia in these cases varies from 24 hours to as many years after the enucleation. A time limit can not be set, but all such cases should be carefully scrutinized clinically and pathologically, before being accepted. The severity of the case under these circumstances may be very great, as witness the present instance. A good summary of the literature is given by Welton. (American Journal Ophthalmology Vol. 40 p. 378.)

2. *The appearance of iritis spongiosa.* From my experience, and from what is given in the description of sympathetic ophthalmia, the usual type of inflammation, as far as the iris is concerned, is the "quiet" type, with a minimum of ciliary injection and pain, usually, in fact with none at all. Attention is called to the eye by dimness of vision. The pupil is small and attempts to dilate it will reveal the fact that synechia have already formed, often of an unyielding character. In this case, a typical iritis spongiosa, with a large lens shaped translucent mass of exudate into the anterior chamber, developed on the 24th day. The mass quickly shrank, but while present it deceived one observer as to its character, being mistaken for a dislocated lens.

3. *The absence of "Descemetitis."* Fuchs lays much stress on the presence of deposits on Descemet's membrane. They were entirely absent in this case until very late, and the cornea was always bright and perfectly clear. The other symptoms would certainly justify the diagnosis of sympathetic ophthalmia.

4. *The occurrence of retinal detachment.* It is very likely that retinal detachment occurs in many cases, but it is usually a terminal condition, when the organization of vitreous exudates, and a partial absorption of the vitreous body, contributes to its production. Under these circumstances, the retinal detachment can not be seen, but can only be inferred.

We know that most eyes that are removed for chronic iridocyclitis will show detachment of the retina. But in this case, the media were fairly clear and the detachment could be readily seen. Moreover it completely disappeared. It behaved more like the retinal detachment of the puerperium. Its appearance was coincident with the deepening of the periphery of the anterior chamber, and it seemed to be due to the same cause, namely a contraction of the cyclitic exudate in the posterior chamber. The restoration of the normal depth of the anterior chamber and the reattachment of the retina indicate that the exudate was absorbed, and the contraction it had occasioned was relieved.

CASE 2. In 1906, I first saw Mr. M., aged then 42. The vision in the right eye was 20/20 not improved by glasses. The left eye was myopic, vision 3/200, with -4.50 — 1.50 cy. ax. 30° , vision 20/40. The fundus was normal. In 1908, the left lens began to be opaque. In October, 1920, the lens was completely opaque and its removal was advised, as the right eye showed both lenticular and vitreous opacities, but practically normal vision. On the morning of Nov. 17, 1920, the lens was extracted from the left eye. The corneal stitch was used, and the extraction was without iridectomy. The lens capsule was grasped and torn with the capsule forceps, and at this time there was the escape of a fluid having the consistency of aqueous, being apparently a very fluid vitreous. On attempting to deliver the lens in the usual way by pressure, it would not present at the wound, and fluid vitreous continued to escape. The lens was removed with a wire loop and the corneal suture tied. As the ball was markedly collapsed, it was filled with saline solution till its normal contour was restored. The saline was introduced by means of a medicine dropper inserted at one corner of the corneal wound. The patient left the table with the eye apparently in a satisfactory condition. After an uneventful day, the patient was attacked with nausea and vomiting at 7 p. m., and again at midnight. The retching was violent and the left eye pained a good deal during and after the attacks. The eye was not dressed till the next morning, when the dressings were found to be blood stained, a clot hung from the inner angle of the corneal wound, and the iris was prolapsed on both sides of the corneal stitch. The anterior chamber was deep, the cornea bright and clear. The corneal stitch was removed at the end of one week. Two weeks after the operation, the prolapse

was flattened and the pupil was filled with a brown mass, apparently blood. There was no reflex obtainable and light perception had not been present since the first dressing. The usual antiphlogistic measures were being employed, and the progress of the case was toward recovery of the eye for six weeks, except for the development of the so-called "atropin conjunctivitis," about five weeks after the operation. On Dec. 27th, about six weeks after the operation, the patient said the *right eye* was uncomfortable, and had been so for 24 hours. There was slight tenderness and ciliary injection, vision 20/40 and pupil active. In consultation, Dr. J. L. Minor interpreted these symptoms just as I did, and agreed to the advice already given to enucleate the left eye, which was done that afternoon. Atropin and hot applications to the right eye with mercurial inunctions and large doses of sodium salicylat, as recommended by Gifford, were begun at once. The pupil dilated widely and evenly, the cornea remained clear, the few vitreous opacities already present showed no increase, though the inflammation, as evidenced by the redness of the eye, was very great. The tenderness did not increase. At no time was there any alteration of the intraocular tension. One week after the onset of these symptoms and the removal of the left eye, Dr. Minor saw the patient again. He called attention to the features of the case in which it was not typical of sympathetic ophthalmia, namely the absence of deposits on Descemet and exudate in the vitreous, the ease with which the pupil dilated and was kept dilated, and the violent inflammatory appearance of the eye, i. e., the redness. Just prior to this consultation, I had obtained from other sources a history of an ancient luetic infection. There had been no treatment for seven or eight years, and the last Wassermann was negative. Salvarsan was added to the treatment and the eye soon began to improve. Three weeks from the onset of the inflammation, the patient was permitted to go out of doors for a ride, and a week later he came to the office for treatment, which was soon stopped. The eye made a complete recovery and is now (June 28th, 1922) normal in every way, except for the lenticular and vitreous opacities which antedated the inflammatory attack. Vision is 20/25—2 with glasses. There was no relapse.

The enucleated eye showed the corneal wound with incarcerated iris, extensive hemorrhage into the ball beneath the choroid, and between the choroid and the retina, and a



Fig. 5. Case 2. Section through the operation wound, which is filled with organized and pigmented tissue, to which the iris is attached. Anterior chamber obliterated below the wound, where the cornea is greatly thickened. Above the wound, cornea of normal thickness. Anterior chamber and iris in normal relation. (x. 15.)

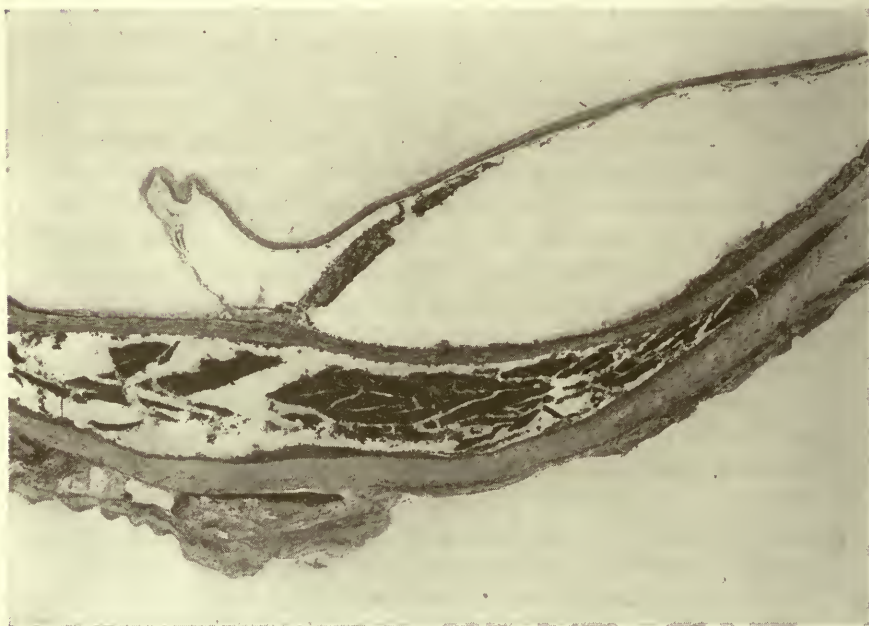


Fig. 6. Case 2. Subretinal and subchoroidal hemorrhage. (x. 10.)



Fig. 7. Case 2. Irregular thickening of the inner layers of the retina, with subretinal hemorrhage. The choroid shows marked nodular infiltration, especially dense at X. (x. 27.)

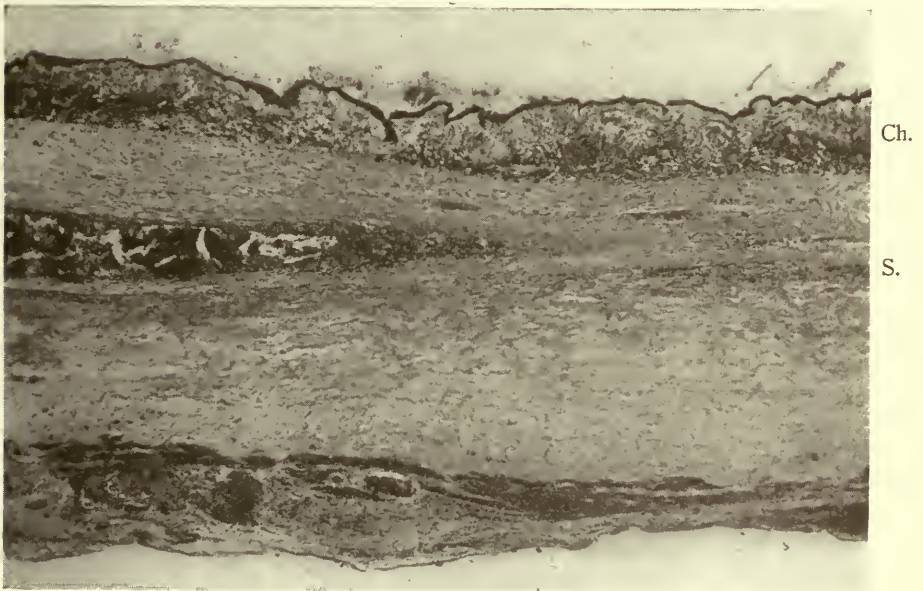


Fig. 8. Case 2. The sclera is split by a hemorrhage between its fibers. The choroid shows the nodular infiltration seen in sympathetic ophthalmia. (x. 36.)

marked inflammatory reaction in the retina, choroid, ciliary body and edges of the corneal wound. Of especial interest are the changes in the choroid. The round celled infiltration shows a decided tendency to a nodular formation, such as is described as the typical change seen in sympathogenic eyes. The ciliary body did not show excessive inflammatory infiltration. Figs. 5, 6, 7, 8.

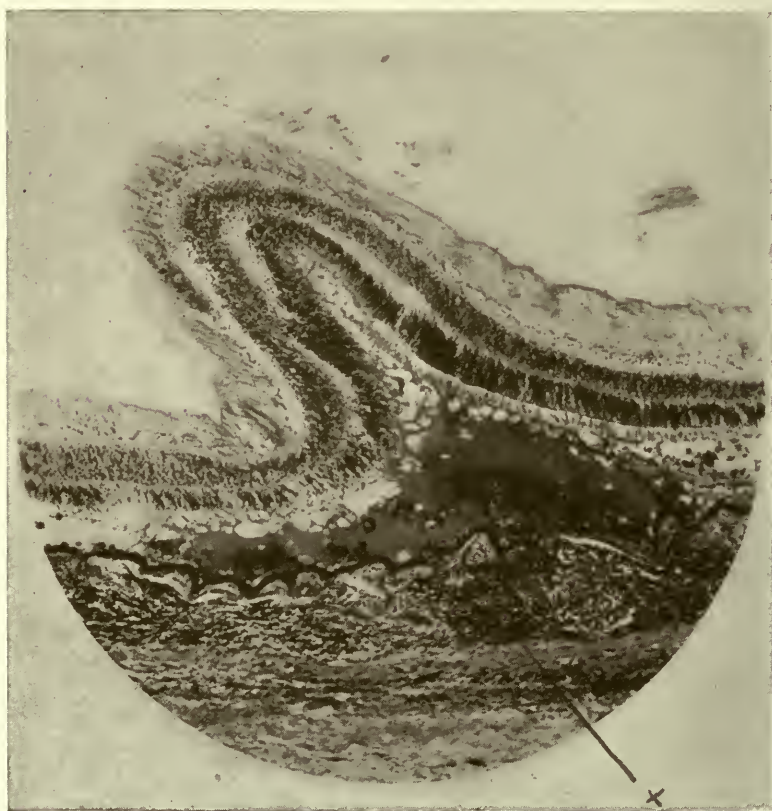


Fig. 9. Case 2. Showing at X a typical nodule of infiltration of the choroid. (x. 60.)

DISCUSSION.

DR. DON M. CAMPBELL, Detroit: It seems to me there is considerable doubt as to whether in this first case he had a case of sympathetic ophthalmia. The examination the patient was subjected to, for the general condition, included the tonsils and accessory sinuses of the nose, and a blood Wassermann. It did not include a tuberculin test, and it did not include focal infections from a great many sources in the body other than those cited in his paper. It is quite possible there may have been a focal infection from the gastrointestinal tract, or chronic appendicitis. So I do not feel that from the data we can say that this individual did not have some other cause for the inflammation which destroyed the second eye.

I would like to have Doctor Ellett also analyze the 17,000 white count in his leucocytosis case. It has been found that the leucocytosis that comes with sympathetic ophthalmia has a very definite character. I refer to the increase in the large mononuclears. That, in my experience, has been a very definite means of determining the threatened advent of a sympathetic ophthalmia, and I think it is interesting to report in this connection, that in those cases of industrial injury where we have a possibility of sympathetic ophthalmia—a watering of the eye and recession of the near point—upon examination it was found that there were ten to fifteen per cent large mononuclear cells. Upon enucleation of the injured eye in these cases, that leucocytosis promptly disappeared. On the other hand, in another case in which a sympathetic ophthalmia followed a cataract operation, done by myself, we found that the condition had advanced a little farther than these subjective symptoms—there was a deep ciliary injection and sometimes a synechia. In that case there was an increased mononuclear count, which, strange to say, did not disappear after enucleation of the eye, nor did the irritation of the sympathizing eye disappear.

The use of salvarsan in sympathetic ophthalmia seems to me is important. The type of blood picture we get in sympathetic ophthalmia very closely simulates that which we find in the infection we know as syphilis. So I think the injection of salvarsan has a real basis for use. In the last case I report, sympathetic ophthalmia following cataract extraction, there certainly has been improvement in the condition.

DR. HARRY S. GRADLE, Chicago: We must be very cautious in defining sympathetic ophthalmia, and our definition is dependent entirely upon our clinical observations. What one man will call sympathetic ophthalmia, or sympathetic neuroretinitis, will not be classed as that at all by another man. We have no definite serologic tests of any type that will allow us to pass upon a case as sympathetic ophthalmia or not. There is, however, the change in the differential blood count. When I first reported that in 1910, I believed it was diagnostic of sympathetic ophthalmia, and attached a great deal of prognostic importance to it; but I have changed my views, and now I believe the blood picture has the same value as the von Pirquet test—it is of negative value. By that I mean that sympathetic ophthalmia will not supervene unless the changes I have described are present; but the presence of changes in the blood does not necessarily mean sympathetic ophthalmia.

In this change there is not an absolute leucocytosis. In fact, the white cells may not be changed in number, but there is a marked change in the relation of the different elements, and considering the increase in the mononuclear cells, both small and large, we are probably justified in calling it a mononucleosis. A mononucleosis is significant, but it is not diagnostic.

The other test of recent origin upon which there has been some development, is the sensitization test of Allen Wood. This is based on the theory that sympathetic ophthalmia is due to sensitization by the uveal pigment. If we believe in that theory—and I for one do—then we will be in position eventually to tell within a short time whether we are dealing with a sympathetic ophthalmia, or with an iridocyclitis due to some other etiology. We cannot depend upon the histologic picture, as was shown in a case I reported. A young man developed a perforating ulcer due to Neisserian infection. The eye was eviscerated by two eminent ophthalmic surgeons. For two years he had no trouble. Then he developed in the other eye a low type of iridocyclitis that failed to yield to treatment. He showed a mononucleosis, and on the strength of that we removed the stump of the eviscerated eye, and in that stump we found a bit of choroidal pigment still left from the evisceration. Histologically, his stump showed the picture described by Fuchs as characteristic of sympathetic ophthalmia. Under treatment with salicylates, the eye cleared. Then about three years later, he developed in his remaining eye an iridocyclitis of exactly the same type—exactly the same picture. But this time we found a 4-plus Wassermann, and upon proper specific treatment the eye cleared up promptly. The first time we thought we were dealing with a sympathetic ophthalmia. But was it that, or was it an iridocyclitis in which we had two elements, one of lues and one of sensitization from the bit of choroidal pigment left? This shows, that until we have some accurate means, we are not justified in calling a case sympathetic ophthalmia unless it is carefully scrutinized for every possible etiologic factor.

DR. J. G. DORSEY, Wichita, Kansas: Schmidt-Rimpler demonstrated that you seldom had sympathetic ophthalmia in people who are not otherwise sick. In the fifth edition of Fuchs, Duane says that Brownlee and others have demonstrated the fact that sympathetic ophthalmia shows some analogy to the protozoal diseases. One of the proven points is the manner in which salvarsan acts in the disease—which is positively. I cannot help but feel, that one of the reasons why mercury is as efficient as it is in these cases, is due to its action on the protozoa.

A peculiar thing in my experience with sympathetic ophthalmia is, that the only two cases that have followed work I have done in our own office have occurred, the first five days after the enucleation of the offending eye, and the second five weeks after enucleation. In the early part of August a case presented itself—a man had been injured with a piece of metal that remained in the eye. Three weeks before this, he had received the injury and had been treated elsewhere, the physician stating that he thought he had removed the foreign body. When he came in, the injured eye was very irritable. The uninjured eye was apparently healthy, the vision 20/20, but the excessive irritability of the injured eye led us to urge that he have it enucleated. The eye healed and the man went home in two weeks, and in five weeks after-

wards presented himself with an iridocyclitis. The case was improving when I left home, but he was not yet well. He was put upon the salicylates and purgatives—quite active purgation I think is advisable in these cases—with atropin, and he was improving.

The other case was one in which there was no rupture of the globe. Since the eye did not heal so well and there was a good deal of edema, we suggested that it be removed, and after some little hesitation this was done. Five days after this, the other eye began to show symptoms of sympathetic ophthalmia.

DR. JOHN A. DONOVAN, Butte, Montana: I want to report a case in line with these. A man had his eye enucleated about four years ago; he did not know there was a ball put in. About three months ago, he began to show signs of sympathetic ophthalmia, and the ophthalmoscope showed some fibrous opacities. He had a vision of 20/50. We put him on the salicylates and sent him to a dentist, who found ten teeth to be taken out. That improved him quite a little, and a couple of weeks later he had his tonsils removed, and he is a good deal better now. I rather think the tonsils were the cause of his trouble, yet if he does not continue to improve I will remove that glass or metal ball, whatever it may be.

DR. E. C. ELLETT, Memphis, Tenn. (closing): In reply to a question raised in the discussion, the differential count made on February 3rd, 1922, was: White cells 11,700, small lymphocytes 8%, large mononuclears 5%, polymorphonuclears neutrophils 87%. Culture from the throat on February 7th showed streptococci and staphylococci.

A STUDY OF THE FUNDUS CHANGES IN NEPHRITIS; SECOND SERIES.*

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In the previous paper¹, the tabulated histories were abstracted from a series of observations made in the clinic of ophthalmic surgery of the University of Michigan. The number of cases then reported was 73. All of the 136 cases studied in this series were derived from the same source, and only those cases are included which have also been examined in the department of internal medicine, the medical findings and diagnoses being derived as in the previous paper from the notes made in that department. The diagnostic classification is based on the clinical manifestations of the disease, rather than on the numerous variations in the histopathologic changes found at autopsy. While there is in most instances a striking similarity in the percentages of the characteristic features, certain interesting variations appear, to which attention will be called in the analysis of the tables. Unfortunately no comparisons of value can be made in acute nephritis or in the miscellaneous cases, as only one of the former and two of the latter came under our observation. These cases, therefore, will not be discussed further than to call attention to the fact, that the two miscellaneous cases were selected because the changes found resembled those frequently seen in nephritis, and the fundus changes in the acute case would suggest investigation of the function of the kidney.

In the tabulation of the cases, the different types have been grouped in five tables, A, B, C, D and E. These tables are then summarized in table 6, where the percentages of the different findings may be easily compared. Graphic chart No. 1 represents the percentages as they would appear in an equal number of cases of each type.

*This is a continuation of the report presented before the Section on Ophthalmology of the American Medical Association, June 1916.

TABLE 1.—SERIES A—CHRONIC INTERSTITIAL NEPHRITIS

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
1. S.I. 2/24/16. Diagnosis: Chronic interstitial nephritis. Urine: Albumin: moderate amount; few hyalin and granular casts; few R.B.C. Blood: B.P. 260 to 220. Orthodiagram, left sided hypertrophy.		M	67	5/7.5	5/60	O.D. Disc blurred; veins partially obscured by "veiling." Disc swollen $\frac{1}{4}$ D. Vessels tortuous; fusiform dilations of veins; endarteritis; arteriovenous compression; perivasculitis; whole retina edematous; hemorrhages in macula; some whitish points in macula; macula edematous. O.S. Disc blurred; marked periarthritis; endarteritis; retina markedly edematous; glistening white dots in macula; macula edematous. Central choroidal changes.
2. A.S. 5/1/16. Diagnosis: Chronic nephritis; arteriosclerosis. Urine: Small amount albumin; few granular casts. Phenolsulphonethalein 25% to 13% in 2 hours. Blood: B.P. 205-162 Hgb. 78%; R.B.C. 4,400,000, W.B.C. 7,600, blood urea, 0.066 gms. per 100 cc. Orthodiagram, left sided hypertrophy.		M	40	5/7.5	5/12	O.D. Disc edematous; veins tortuous; arteries small; macula granular; pigment change in macula and 2 disc diameters from macula. O.S. Disc edematous; arteries contracted; veins finely tortuous; retina edematous; macula edematous and granular.
3. A.S. 5/15/16. Diagnosis: Arteriosclerotic nephritis; chronic hypertensive arteriosclerosis; cardiac insufficiency. Urine: No albumin; few granular casts; many W.B.C.; phenolphthalein 33% in 2 hours. Blood: B.P. 170-172; Hgb. 75%; R.B.C. 3,500,000, W.B.C. 7,200.		M	68	6/10	6/7.5	O.D. Retina edematous; slight diffuse chorioretinitis throughout. O.S. Disc edematous; veins engorged; periarthritis; chorioretinitis and edema; macula hyperemic and granular.
4. M.F. 7/2/16. Diagnosis: Chronic interstitial nephritis; hypertension; arteriosclerosis. Urine: No albumin; no casts; phenolphthalein, 45% in 2 hours. 190-230; Hgb. 85%; R.B.C. 4,670,000; W.B.C. 10,800; blood urea, 0.036 gms. per 100 cc.		F	47	6/10	6/5	O.D. Disc and retina edematous; hemorrhages; marked arteriovenous compression; contracted arteries; flocculent like exudates; macula edematous, radiating exudates in macula; endarteritis. O.S. Disc edematous; arteries contracted; marked arteriovenous compression; veins irregular, fusiform dilations along arteries; macula edematous and granular.
5. E.P. 7/14/16. Diagnosis: Chronic interstitial nephritis; hypertension; arteriosclerosis. Blood: B.P. 235; Hgb. 98%; R.B.C. 4,880,000, W.B.C. 7,750; blood urea 0.028 gms. per 100 cc.; blood Wassermann plus-minus. Urine: No albumin; no casts; phenolphthalein 36% in 2 hours, and later 57% in 2 hours.		M	58	5/30	5/30	O.D. Disc hyperemic and edematous; arteries attenuated; copper wire vessels; irregular vessels. Veins engorged; marked arteriovenous compression; retina edematous; numerous dull white spots scattered through fundus, apparently deep retinal; macula shows number of large white spots. O.S. Small deposit of pigment on disc; arteries almost obliterated in places. Otherwise same as O.D.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
6.	C.W. 9/13/16..... Diagnosis: Chronic interstitial nephritis. Urine: Small amount of albumin; few R.B.C.; few casts. B.P. 165; Hgb. 55%; R.B.C. 3,580,000; W.B.C. 9,200; blood urea 0.135 gms. per 100 cc.; blood urea (4 days later) 0.110 gms. per 100 cc.; blood creatinin 4.25 mg. per 100 cc.	M	53	5/12-1	5/12	O.D. Deep cupping, resembling glaucoma; arteriovenous compression; retina slightly edematous. O.S. Rings blurred; arteries small, otherwise same as O.D.
7	G.A. 11/2/16..... Diagnosis: Chronic interstitial nephritis; tabes dorsalis. Urine: No albumin; no casts. Blood: B.P. 150-245; Hgb. 85%; R.B.C. 5,200,000; W.B.C. 15,250; blood urea 0.144 to 0.042 gms. per 100 cc.; blood Wassermann, plus-minus. Spinal fluid: Slight increased pressure; carbolic +; ammon. sulph. +; albumin increased; cell count 20 per cu. mm.; Wassermann +++.	M	51	5/10-1	5/12-2	O.D. Cup excavated; lamina cribrosa somewhat edematous; marked arteriovenous compression; arterial walls thickened; endarteritis; veins irregular; arteries irregular; retina edematous. O.S. Macular region edematous. Otherwise same as O.D.
8.	C.S. 12/5/16..... Diagnosis: Arteriosclerosis. Urine: No albumin; no casts; few R.B.C.; phenolphthalein 57% in 2 hours. Blood: B.P. 190; Hgb. 83%; W.B.C. 6,400; blood urea 0.035 gms. per 100 cc; blood Wassermann negative. Orthodiagram, area 156%.	M	64	5/15	5/15	O.D. Disc edematous, blurred; arteries small; veins engorged and irregular; arteriovenous compression; thinning of retinal epithelium and pigment. O.S. Marked sclerosis of choroidal vessels. Endarteritis. Otherwise same as O.D.
9.	J.S. 4/17/17..... Diagnosis: Chronic interstitial nephritis; hypertension. Urine: Small amount albumin; few casts; phenolphthalein 35% in 2 hours. Blood: B.P. 180; Hgb. 95%; R.B.C. 4,700,000, W.B.C. 8,500; blood urea, 0.034 gms. per 100 cc. Orthodiagram, area 103%.	M	72	?	?	O.D. Disc edematous; rings blurred; arteries markedly reduced in caliber; endarteritis; copper wire arteries; arteriovenous compression marked; cork-screw vessels; tortuous veins; central retinitis, composed of collection of yellowish dots. O.S. Arteriosclerotic changes more marked; otherwise same as O.D.
10.	J.M. 7/20/17..... Diagnosis: Chronic interstitial nephritis; hypertension. Urine: Small amount albumin; many hyalin and granular casts. Blood: B.P. 196 to 240; Hgb. 90%; W.B.C. 17,000; blood Wassermann, plus-minus. Spinal fluid: Negative.	M	42	?	?	O.D. Rings blurred; disc hyperemic; arteries reduced in caliber, some beaded and tortuous; veins beaded and tortuous; some venous engorgement; choroidal vessels conspicuous. O.S. Some exudate on floor of cup; disc pale; some hemorrhages; marked endarteritis; large grayish-white patch of exudate (several); otherwise same as O.D.

11.	M.C. 11/19/17..... Diagnosis: Chronic interstitial nephritis; hypertension. Urine: No albumin; few R.B.C.; few granular casts; many W.B.C. Blood: B.P. 155 to 172; Hgb. 95%; W.B.C. 7,200; blood urea 0.045 to 0.048 gms. per 100 cc.	F	56	5/60	5/60	O.D. Nerve head slightly edematous; rings blurred; arteries reduced in caliber; veins engorged and tortuous; retina slightly edematous; macula shows marked edema with definite punctate disturbance; choroidal changes. O.S. Nerve head edematous; there is a small grayish-white spot in retina just temporally from disc margin; moderate arteriovenous compression; several white dots in macular region; arteries are copper wire like.
12.	A.S. 3/13/19..... Diagnosis: Chronic interstitial nephritis; hypertension; arteriosclerosis. Urine: Small amount of albumin; few granular casts. Orthodiagram, area 176%. Blood: (Not reported.)	F	59	L.P.	L.P.	O.D. Cupping of disc; arteries markedly contracted; some proliferation of pigment throughout fundus; choroidal change below; several areas of atrophic change; retina atrophic throughout. O.S. Rings blurred; arteries contracted; veins tortuous; atrophic changes as in O.D.; several yellowish dots in macula, disc edematous.
13.	D.G. 4/9/19..... Diagnosis: Chronic interstitial nephritis; hypertension. Urine: Small amount albumin; few granular and hyalin casts; phenolphthalein 30% in 2 hours. Blood: B.P. 190; Hgb. 75%; R.B.C. 3,650,000, W.B.C. 5,800; blood urea 0.072 gms. per 100 cc. Orthodiagram, area 128%.	M	53	C.F. at 1 ft.	5/7.5-1	O.D. Rings blurred; numerous very fine and fusiform hemorrhages throughout; retina very edematous; many hemorrhages of all sizes and shapes; perivasculitis; macular area surrounded and included in large hemorrhage; well marked endarteritis; marked arteriovenous compression; veins irregular; arteries contracted. O.S. Silver wire arteries in macular region; otherwise same as O.D. except for only one large, deep, absorbed hemorrhage.
14.	L.C. 4/23/19..... Diagnosis: Chronic interstitial nephritis; arteriosclerosis; hypertension. Urine: Moderate amount albumin; few granular casts; phenolphthalein 40% in 3 hours. Blood: B.P. 210; Hgb. 95%; W.B.C. 13,100; blood urea 0.065 gms. per 100 cc.; blood sugar 0.130%; blood Wassermann negative.	M	58	?	?	O.D. Nerve head edematous; arteries contracted; arteriosclerosis. O.S. Rings blurred; some of arteries almost obliterated; marked arterial sclerosis.
15.	D.B. 8/18/19..... Diagnosis: Chronic interstitial nephritis; hypertension. Urine: Trace of albumin; few hyalin casts; few W.B.C.; phenolphthalein 6% in 2 hours. Blood: B.P. 280; Hgb. 70%; R.B.C. 4,100,000, W.B.C. 9,600; blood urea 0.044 gms. per 100 cc.	F	57	C.F. at 2 ft.	10/200	O.D. Nerve head markedly edematous; rings blurred; veins engorged; marked arteriovenous compression; large hemorrhage; fine areas scattered about macula; retina edematous. O.S. Few small hemorrhages; otherwise same as O.D.
16.	S.C. 3/19/20..... Diagnosis: Chronic nephritis; myocarditis. Urine: Small amount albumin; many granular casts; few W.B.C.; phenolphthalein 40% in 2 hours. Blood: B.P. 130 to 135; Hgb. 85%; W.B.C. 7,300; blood urea 0.051 gms. per 100 cc.	F	57	5/20	5/10	O.D. Rings blurred; veins engorged and tortuous; arteriovenous compression. O.S. Arteries contracted; veins engorged.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
17.	I.D. 5/4/20. Diagnosis: Chronic nephritis; hypertension. Urine: No albumin; few granular casts; many W.B.C.; phenolphthalein 40% in 2 hours. Blood: B.P. 150 to 215; Hgb. 100%; W.B.C. 7,500; blood urea 0.023 to 0.035 gms. per 100 cc.; Wassermann negative.	F	59	5/20-1	5/20	O.D. Veins engorged; arteries irregular; endarteritis; perivasculitis; arteriovenous compression; retina slightly edematous; some of macular veins show corkscrew characteristics. O.S. Slight edema of disc and retina; variation in caliber of arteries; otherwise same as O.D.
18.	W.H. 10/26/20. Diagnosis: Chronic nephritis. Urine: No albumin; no casts; many W.B.C.; phenolphthalein 62% in 2 hours. Blood: B.P. 180 to 195; Hgb. 96%; R.B.C. 4,990,000; W.B.C. 7,100; blood urea 0.006 gms. per 100 cc. Orthodiagram, area 130%.	M	66	5/7.5-2	5/6	O.D. Disc edematous; edema and loss of transparency of retina; arteries irregular and contracted, some nearly wire like; bright dot along superior nasal artery; veins markedly tortuous, almost corkscrew like; endarteritis; macula edematous. O.S. Rings blurred; marked arteriovenous compression; otherwise same as O.D.
19.	F.A. 11/21/20. Diagnosis: Chronic interstitial nephritis; hypertension. Urine: Small amount albumin; few granular casts; phenolphthalein 38% in 2 hours. Blood: B.P. 220 to 230; Hgb. 95%; R.B.C. 5,240,000; W.B.C. 9,000; blood urea 0.031 to 0.045 gms. per 100 cc.	F	56	5/10	5/12	O.D. Disc edematous; rings blurred; veins engorged and tortuous; vessels irregular in caliber; arteries contracted; perivasculitis; endarteritis; macula edematous. O.S. Arteriovenous compression. Otherwise same as O.D.
20.	S.B. 1/16/21. Diagnosis: Chronic interstitial nephritis. Urine: No albumin; few W.B.C. no casts; phenolphthalein 35% in 2 hours. Blood: B.P. 120; Hgb. 90%; R.B.C. 4,500,000, W.B.C. 8,300; blood urea 0.026 gms. per 100 cc.; blood Wassermann, negative. Orthodiagram, 77%.	F	58	5/12	5/20	O.D. Endarteritis; arteriovenous compression; marked retinal edema and edema of disc; some arteries are coppery in appearance, macular vessels tortuous; macula edematous. O.S. Same as O.D.
21.	S.S. 1/19/21. Diagnosis: Chronic nephritis; arteriosclerosis; hypertension chronic myocarditis. Urine: Small amount of albumin; many W.B.C.; occasional hyalin cast; phenolphthalein 55% in 2 hours. Blood: B.P. 185 to 200; Hgb. 80%; R.B.C. 4,360,000; W.B.C. 10,800; blood urea 0.039 gms. per 100 cc.	F	61	5/6+	5/6+	O.D. Rings blurred, edematous; loss of transparency of retina; retina edematous; endarteritis; veins engorged; arteriovenous compression. O.S. Hemorrhage; infiltration of retina, grayish creamy color; macular veins tortuous, some corkscrew like; otherwise same as O.D.

TABLE 2.—SERIES B—HYPERTENSION

		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
TABLE 2.—SERIES B—HYPERTENSION						
Medical Findings and Diagnosis						
22.	L.D. 4/21/21. Diagnosis: Chronic nephritis; hypertension. Urine: Small amount of albumin; few casts; many W.B.C. 15; Hgb. 56% in 2 hours. Blood: B.P. 165; Hgb. 95%; R.B.C. 5,350,000; W.B.C. 10,500; blood urea 0.086 gms. per 100 cc. Orthodiagram, area 131%.	F	59	5/30+1	5/30+1	O.D. Rings blurred; arteries reduced in caliber; veins engorged; small hemorrhage; arteriovenous compression. Some yellowish spots between macula and disc. O.S. Marked arteriovenous compression; veins engorged; edema of retina.
23.	E.D. 11/21/21. Diagnosis: Chronic nephritis. Urine: Small amount albumin; few W.B.C.; no casts; phenolsulphonphthalein 55% in 2 hours. Blood: B.P. 182 to 185; Hgb. 108%; R.B.C. 4,820,000; W.B.C. 6,400; blood Wassermann negative. Orthodiagram, area 121%.	M	64	5/7.5 + 3	5/6-2	O.D. Disc hyperemic; margins blurred; veins full; arteries small and contracted; arteriovenous compression; some variation in caliber of arteries; macular region granular and edematous; few indistinct whitish patches in macula. O.S. Same as O.D.
Medical Findings and Diagnosis						
1.	A.K. 2/24/16. Diagnosis: Hypertension. Urine: No albumin; occasional granular and few hyalin casts; few W.B.C.; phenolphthalein, 47% in 2 hours. Blood: B.P. 212 to 264; Hgb. 85%; R.B.C. 5,000,000; W.B.C. 7,300 to 9,300. Orthodiagram, cardiac hypertrophy and dilation.	F	49	2/60	5/4	O.D. No fundus details (occluded pupil). O.S. Disc blurred, congested and edematous; rings blurred; arteries contracted; arteriovenous compression; veins tortuous; retina edematous; macula edematous.
2.	E.D. 11/15/16. Diagnosis: Hypertension; arteriosclerosis. Urine: No albumin; no casts; few W.B.C.; phenolphthalein 44% in 2 hours. Blood: B.P. 175; Hgb. 91%; W.B.C. 10,550; blood urea 0.037 gms. per 100 cc.	F	64	5/7.5-2	5/6-1	O.D. Nerve head hyperemic and edematous; rings blurred; arteries attenuated; veins engorged and tortuous; slight arteriovenous compression; retina very edematous; perivascularitis; copper wire arteries; endarteritis. O.S. Same as O.D.
3.	M.M. 5/31/17. Diagnosis: Chronic nephritis; hypertension. Urine: No albumin; occasional hyalin cast. Blood: B.P. 210 to 220; Hgb. 75%; R.B.C. 4,300,000; W.B.C. 7,300; blood urea 0.055 to 0.062 gms. per 100 cc. Orthodiagram, area 145%.	M	56	5/60	5/60	O.D. Disc hyperemic, edematous; veins contracted; arteriovenous compression; retina very edematous; small hemorrhage over disc; small spot of black pigment below disc; macula very edematous. O.S. Medullary sheathitis. Otherwise same as O.D.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
4.	L.R. 11/24/17..... Diagnosis: Hypertension. Urine: Small amount of albumin; few hyalin casts; many R.B.C. and W.B.C.; phenolphthalein 40% in 2 hours. Blood: B.P. 180 to 220; Hgb. 103%; R.B.C. 6,760,000, W.B.C. 4,400; blood urea 0.055 gms. per 100 cc. Orthodiagram, area 128%.	F	53	5/20	5/15	O.D. Nervehead slightly edematous; reflex stripe marked. O.S. Nervehead slightly edematous; arteriovenous compression.
5.	A.T. 12/17/17..... Diagnosis: Hypertension. Urine: No albumin; few hyalin casts; phenolphthalein 46% in 2 hours. Blood: B.P. 185; Hgb. 63 and 56%; R.B.C. 3,600,000 to 4,100,000, W.B.C. 9,300; blood urea 0.034 gms. per 100 cc. Wassermann negative.	M	53	5/5-3	5/30	O.D. Nervehead blurred; rings blurred; arteries small; marked arteriovenous compression. O.S. Same as O.D.
6.	B. de P. 3/6/18..... Diagnosis: Chronic nephritis; circulatory failure. Urine: Small amount of albumin; no casts; phthalein 52% in 2 hours. Blood: B.P. 210; Hgb. 86%; R.B.C. 4,560,000, W.B.C. 6,600.	F	42	5/5-3	5/5	O.D. Vessels tortuous and irregular; veins engorged; arteries copper wire like; macula granular. O.S. Arteriovenous compression; otherwise same as O.D.
7.	M.D. 3/18/18..... Diagnosis: Hypertension. Urine: No albumin; few granular casts; few W.B.C. Blood: B.P. 180 to 240; Hgb. 95%; W.B.C. 6,550; blood urea 0.048 gms. per 100 cc.	F	60	?	?	O.D. Disc pale; vessels reduced in caliber; tortuous and beaded; arteriovenous compression. O.S. Same as O.D.
8.	H.B. 12/19/18..... Diagnosis: Chronic nephritis; hypertension. Urine: Trace of albumin; few granular and hyalin casts; few W.B.C. phthalein 43% in 2 hours. Blood: B.P. 170 to 180; Hgb. 80%; R.B.C. 4,000,000, W.B.C. 6,700; blood urea 0.034 to 0.037 gms. per 100 cc.; Wassermann negative.	F	61	5/30	5/30	O.D. Nervehead hyperemic, edematous; arteriovenous compression; endarteritis; veins are engorged; macular vessels tortuous. O.S. Slight edema of retina; macula granular; otherwise same as O.D.
9.	S.H. 2/23/19..... Diagnosis: Hypertension; arteriosclerosis. Urine: No albumin; no casts; many W.B.C.; phthalein 50% in 2 hours. Blood: B.P. 205; Hgb. 90%; W.B.C. 6,500; blood urea 0.040 per 100 cc.	F	71	5/30	L.P.	O.D. Nervehead hyperemic and edematous; rings blurred; arteriovenous compression; endarteritis; arteries contracted; old central choroiditis. O.S. Same as O.D.

10.	I.B. 7/11/19. Diagnosis: Chronic nephritis; hypertension. Urine: No albumin; no casts. Blood: B.P. 180 to 190; Hgb. 80%; R.B.C. 3,600,000; W.B.C. 5,600; blood urea 0.025 gms. per 100 cc; Wassermann negative.	F	62	5/60	5/15+	O.D. Disc edematous; rings blurred; arteries small; endarteritis; veins engorged and tortuous; vessels corkscrew like; many hemorrhages near disc and in macula, some flame shaped; retina edematous; numerous hemorrhages throughout; pigment changes. O.S. Arteriovenous compression; numerous yellowish white spots along macular vessels, between disc and macula; some pigment changes; otherwise same as O.D.
11.	A.H.A. 9/10/19. Diagnosis: Hypertension. Urine: No albumin; few hyalin casts; few W.B.C.; pathalein 52% in 2 hours. Blood: B.P. 158 to 200, Hgb. 75%; R.B.C. 4,920,000, W.B.C. 5,000.	M	47	6/5-1	6/5	O.D. Rings edematous; arteries reduced in caliber; endarteritis; veins tortuous; macular vessels largely obliterated; yellowish white spots in macula; old hemorrhages around macula; retina edematous; periarteritis; phlebitis. O.S. Same as O.D.
12.	D.N. 10/4/19. Diagnosis: Nephritis hypertension Urine: Trace of albumin; phenolphthalein 63% in 2 hours. Blood: B.P. 220 to 225; Hgb. 95%; R.B.C. 4,880,000, W.B.C. 11,000; blood urea 0.030 gms. per 100 cc.	M	48	5/5	5/7.5	O.D. Disc edematous; arteries reduced in caliber; veins large and show beading; whitish spots along line of superior nasal vein; macula edematous; some very fine whitish spots in macula. O.S. Retina edematous; otherwise same as O.D.
13.	M.H. 12/24/19. Diagnosis: Chronic nephritis; hypertension; arteriosclerosis. Urine: No albumin; no casts; phenolphthalein 35 to 40% in first hour. Blood: B.P. 210 to 250; Hgb. 90%; W.B.C. 5,400; blood urea 0.070 to 0.072 gms. per 100 cc; Wassermann negative.	F	52	5/20-1	5/13	O.D. Rings blurred; arteries small, tortuous; veins engorged; arteriovenous compression; few fine yellowish dots in macular area; retina edematous. O.S. Same as O.D.
14.	P.B. 7/27/20. Diagnosis: Hypertension. Urine: Marked albumin at first, which all disappeared after losing edema; some hyalin casts; phenolphthalein 34% in 2 hours. Blood: B.P. 160 to 200; Hgb. 90%; R.B.C. 4,700,000, W.B.C. 9,400; blood urea 0.014 gms. per 100 cc.; Wassermann negative. Orthodiagram area 124%	M	53	5/10+3	5/5-2	O.D. Rings blurred; vessels engorged; arteriovenous compression; retina edematous; macula edematous. O.S. Same as O.D.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
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15.	A.McG. 1/19/21..... Diagnosis: Hypertension..... Urine: Moderate amount of albumin; few W.B.C.; few R.B.C.; many granular casts; phenolsulphonphthalein 50% in 2 hours. Blood: B.P. 210 to 250; Hgb. 96%; R.B.C. 4,650,000; W.B.C. 5,400; blood urea 0.0336 gms. per 100 cc. Orthodiagram, area 118%;	M	49	5/10	5/10	O.D. Disc slightly edematous; endarteritis; moderate arteriovenous compression. O.S. Disc and retina edematous; arteries small; slight hyalin disturbance in macula; macula granular.
16.	L.T. 2/25/21..... Diagnosis: Hypertension; chronic nephritis; arteriosclerosis. Urine: Trace of albumin; many W.B.C.; phenolsulphonphthalein (not done). Blood: B.P. 180 to 250; Hgb. 100%; R.B.C. 4,800,000; W.B.C. 12,000; blood urea 0.048 gms. per 100 cc.; blood Wassermann negative; spinal fluid Wassermann negative. Orthodiagram, area 107%.	F	46	3/30	3/30	O.D. Arteries small, somewhat irregular; endarteritis; periarteritis; arteriovenous compression; veins full; edema of retina; numerous small mottlings in macula. O.S. Veins engorged; punctate stippled appearance of macula. Otherwise same as O.D.
17.	C.H.M. 7/15/21..... Diagnosis: Hypertension; chronic nephritis. Urine: Trace of albumin; no casts; phenolsulphonphthalein 55% in 2 hours. Blood: B.P. 160 to 200; Hgb. 85%; R.B.C. 4,240,000; W.B.C. 9,100. Orthodiagram, area 153%.	M	37	5/5+	5/6	O.D. Rings blurred; macula slightly hyperemic. O.S. Slight hyperemia.
18.	L.M. 1/5/22..... Diagnosis: Hypertension. Urine: Trace of albumin; few W.B.C.; phenolsulphonphthalein 70% in 2 hours. Blood: B.P. 200 to 208; Hgb. 92%; R.B.C. 5,190,000; W.B.C. 7,800; nonprotein nitrogen 0.024 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 160%.	F	53	5/10-1	5/12	O.D. Disc hyperemic; rings completely blurred; disc swollen; dioptrics, vessels engorged and tortuous; endarteritis; arteriovenous compression; many corkscrew vessels; silver wire arteries; retina marked; macular region granular and tudded with minute white spots; one hemorrhage in macula. O.S. Less swelling of disc; disc blurred; small hemorrhage; same as O.D.
19.	I.S. 1/13/22..... Diagnosis: Hypertension; diabetes mellitus. Urine: No albumin; small amount of sugar; no casts; phenolsulphonphthalein 50% in 2 hours. Blood: B.P. 150 to 170; Hgb. 98%; R.B.C. 4,850,000; W.B.C. 7,500; blood sugar 0.20 to 0.24; blood Wassermann negative. Orthodiagram, area 101%.	F	65	5/10-2	5/15-1	O.D. Disc hyperemic; edematous; endarteritis; arteries are coppery in appearance and irregular in caliber; arteriovenous compression; veins engorged; retina edematous; macula edematous. O.S. Perivasculitis; otherwise same as O.D.

TABLE 3.—SERIES C—CHRONIC PARENCHYMATOUS NEPHRITIS

F	65	5/12-1	5/20-1	O.D.	Rings blurred; disc hyperemic; endarteritis; arterio-venous compression; veins engorged; arteries and veins tortuous; retina edematous. Same as O.D.
H.E. 5/27/22.					
Diagnosis: Arteriosclerosis; chronic nephritis; hypertension.					
Urine: Small amount of albumin; few W.B.C.; few casts; phenolphthalein 33% in 2 hours.					
Blood: B.P. 188 to 215; Hgb. 100%; R.B.C. 4,070,000, W.B.C. 8,100; Nonprotein nitrogen 0.030 gms. per 100 cc.					
Orthodiagram, area 111%.					

TABLE 3.—SERIES C—CHRONIC PARENCHYMATOUS NEPHRITIS

Medical Findings and Diagnosis	Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
1. J.McG. 4/18/16. Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; occasional hyalin and granular cast; small amount of pus. Blood: B.P. 176 to 190; Hgb. 65%; R.B.C. 3,330,000, W.B.C. 9,900; blood urea 0.099 gms. per 100 cc. Orthodiagram, bilateral dilatation.	M	53	5/10	5/10	O.D. Nervehead edematous; arteries attenuated; veins attenuated and irregular; retina edematous; macula edematous; vessels throughout fundus are buried in edema. O.S. Moderate sized diffuse hemorrhage; otherwise same as O.D.
2. A.H. 5/23/16. Diagnosis: Nephritis; hypertension. Urine: No albumin; few R.B.C. and W.B.C.; few hyalin casts; phenolphthalein 50% in 2 hours. Blood: B.P. 142 to 172; Hgb. 85%; R.B.C. 5,550,000, W.B.C. 7,500.	F	39	5/4	5/4	O.D. Nervehead hyperemic, perivasculitis of disc vessels; veins engorged; endarteritis; marked arteriovenous compression. O.S. Rings blurred; disc hyperemic; nervehead edematous; marked venous engorgement; marked arterio-venous compression; arteries tortuous; macular region is finely granular; several distinct yellowish spots near macula apparently in choroid; retina and choroid edematous throughout.
3. J.McQ. 8/29/16. Diagnosis: Nephritis; arteriosclerosis; hypertension; glycosuria. Urine: Trace of albumin; many granular casts. Blood: Blood urea 0.042 to 0.072 gms. per 100 cc.; Hgb. 110%; R.B.C. 6,624,000, W.B.C. 15,200.	M	56	5/30-1	5/20	O.D. Disc hyperemic and edematous; rings blurred; arteries tortuous; marked arteriovenous compression; veins engorged; retina edematous; large area of glistering white, punched out spots above macula and apparently in retina; 2 hemorrhages. Same as O.D. O.S. Same as O.D.

Medical Findings and Diagnosis			Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
4. L.H. 10/6/16. Diagnosis: Chronic parenchymatous nephritis; hypertension. Large amount of albumin; hyalin and granular casts phenolphthalein 0% in 2 hours. Blood: B.P. 200 to 260; Hgb. 55%; R.B.C. 3,750,000; W.B.C. 16,150; blood sugar 1.405 to 1.904 mg. per cc.; blood urea 0.021 to 0.043 gms. per 100 cc. Patient died in uremic coma.			M	51	5/6	5/5	O.D. Nervehead blurred; rings blurred; arteries are irregular and reduced in caliber and copper wire; marked arteriovenous compression; veins engorged and tortuous; retina edematous; number of scattered hemorrhages throughout; number grayish-white areas throughout fundus; large yellowish-white pink spot in macula; macula granular; number of hyalin dots. O.S. Nervehead congested; many old and recent hemorrhages; large bluish-white spot in retina; an old hemorrhage; in macular region, group of glistening grayish spots having tendency to radial arrangement around macula.
5. W.L. 11/22/16. Diagnosis: Chronic nephritis; cerebral arteriosclerosis. Urine: Small amount of albumin; few W.B.C.; few hyalin and granular casts; phenolsulphonphthalein 18% in 2 hours. Blood: B.P. 250 to 260; Hgb. 79%; R.B.C. 5,230,000; W.B.C. 13,500 to 23,100; blood urea 0.024 to 0.049 gms. per 100 cc. Patient died. Autopsy report: Generalized arteriosclerosis; chronic parenchymatous nephritis; syphilitic myocarditis; dissecting aneurysm of aorta.			M	58	?	?	O.D. Nervehead edematous; arteries small, coppery; perivascularitis; endarteritis; veins tortuous and markedly engorged; extreme arteriovenous compression; retina markedly edematous; extreme sclerosis of choroidal vessels; marked central macular change. O.S. Thinning and atrophy of retina throughout; otherwise same as O.D.
6. F.L. 11/25/16. Diagnosis: Chronic nephritis; diabetes mellitus. Urine: Small amount albumin and sugar; few hyalin and granular casts; few W.B.C. and R.B.C.; phenolphthalein 84% in 2 hours. Blood: B.P. 162; Hgb. 88%; W.B.C. 9,200; blood urea 0.030 gms. per 100 cc.; blood sugar 0.114 to 0.171%; blood Wassermann negative; spinal fluid negative.			F	42	5/5-1	5/5	O.D. Nervehead congested and edematous; rings blurred; arteries reduced in caliber; endarteritis; arteriovenous compression; retina congested and edematous; number of white glistening spots in macula. O.S. Pigment disturbance throughout whole choroid. Otherwise same as O.D.
7. L.M. 12/9/16. Diagnosis: Chronic nephritis. Urine: No albumin; sediment shows hyalin and granular casts; phenolphthalein 4% in 2 hours. Blood: B.P. 190; Hgb. 84%; R.B.C. 5,950,000; W.B.C. 9,250; blood urea 0.032 gms. per 100 cc. Orthodiagram, moderate general enlargement; hypertrophy and beginning dilatation.			M	87	5/12-1	5/7.5	O.D. Nervehead edematous; copper wire arteries and reduced in caliber; veins tortuous and engorged; extreme arteriovenous compression; many white glistening spots near disc in retina; retina edematous and in places is atrophic; one macular vessel is silver wire in appearance. O.S. Nervehead hyperemic and edematous; numerous hemorrhages in retina; otherwise same as O.D.

8. S.E. 1/5/17. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few hyalin and granular casts; few W.B.C.; phenolphthalein 32% in 2 hours. Blood: B.P. 195 to 275; Hgb. 70%; R.B.C. 3,800,000; W.B.C. 6,420; blood urea 0.064 to 0.229 gms. per 100 cc. Orthodiagram, area 208%.	M	59	5/30-1	5/30-1	O.D. Rings blurred; nervehead hyperemic and edematous; arteries irregular; endarteritis; perivascularitis; periphlebitis; marked arteriovenous compression; retina hyperemic; choroid granular; remnants of old hemorrhages; small flame shaped and rounded hemorrhages. O.S. Sclerosed choroidal vessels. Otherwise same as O.D.
9. M.S. 1/9/17. Diagnosis: Lues III; chronic nephritis; diabetes mellitus. Urine: No albumin; large amount of sugar; many R.B.C. and W.B.C. no casts. Blood: B.P. 120; Hgb. 75%; W.B.C. 9,740 to 14,500; blood urea 0.051 to 0.054 gms. per 100 cc.; blood sugar 0.228.	M	55	5/10-2	5/10-1	O.D. Rings blurred; veins engorged; marked arteriovenous compression; few small hemorrhages near macula; many more small hemorrhages throughout fundus; retina hyperemic and edematous; endarteritis. O.S. Perivascularitis; otherwise same as O.D.
10. I.F. 3/1/17. Diagnosis: Chronic nephritis (early). Urine: Large amount of albumin; few hyalin and granular casts; an occasional R.B.C. and W.B.C.; phthalein 44% in 2 hours. Blood: B.P. 128 to 132; Hgb. 70%; R.B.C. 3,730,000; W.B.C. 8,600; blood urea 0.037 to 0.073 gms. per 100 cc.	M	31	5/15	5/12	O.D. Rings blurred; veins slightly engorged; slight hyperemia and edema of retina; macula granular. O.S. Nervehead edematous; veins slightly engorged; faint arteriovenous compression; retina slightly edematous; macula very granular.
11. M.E. 4/19/17. Diagnosis: C.N.S. lues; nephritis. Urine: Trace of albumin; few granular casts; few R.B.C. and W.B.C.; phenolphthalein 52% in 2 hrs. Blood: B.P. 180 to 220; Hgb. 82%; R.B.C. 4,320,000; W.B.C. 5,600.	F	57	5/4	5/4	O.D. Disc blurred; rings blurred; arteriovenous compression; macula granular. O.S. Disc hyperemic; veins engorged; retina slightly edematous; macula granular.
12. G.S. 6/21/17. Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; many granular casts; phenolphthalein 20% to 30% in 2 hours. Blood: B.P. 200 to 203; Hgb. 68%; W.B.C. 10,800; blood urea 0.0276 to 0.072 gms. per 100 cc.	F	25	?	?	O.D. Disc hyperemic and edematous; ring blurred; arteries and veins tortuous; arteries copper wire; slight venous engorgement; marked arteriovenous compression. O.S. Same as O.D.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
13. C.H. 8/25/17. Diagnosis: Chronic nephritis; hypertension. Urine: Moderate amount of albumin; many casts; occasional R.B.C. and W.B.C.; phenolphthalein 10% in 2 hours. Blood: B.P. 155 to 175; Hgb. 54%; R.B.C. 3,100,000; W.B.C. 9,150; blood urea 0.072 to 0.200 gms. per 100 cc.		M	50	?	?	O.D. Rings blurred; nervehead edematous; vessels reduced in caliber and tortuous; veins engorged; many large and small granules of grayish exudate scattered throughout fundus; retina edematous; macula granular. O.S. Same as O.D.
14. C.S. 9/29/17. Diagnosis: Chronic nephritis. Urine: Small amount albumin; few hyalin and granular casts; phenolphthalein 0% in 2 hours. Blood: B.P. 195 to 254; Hgb. 35%; R.B.C. 2,840,000; W.B.C. 11,000; blood urea 0.192 to 0.270 gms. per 100 cc.		M	59	?	?	O.D. Normal fundus. O.S. Normal fundus, except for 2 small patches of exudate; disc slightly edematous.
15. B.M. 12/5/17. Diagnosis: Chronic nephritis; hypertension. Urine: Small amount of albumin; few hyalin and granular casts; few R.B.C. and W.B.C.; phenolphthalein 25% in 2 hours. Blood: B.P. 250 to 275; Hgb. 58%; R.B.C. 5,200,000; W.B.C. 9,800 to 14,800; blood urea 0.060 to 0.072 gms. per 100 cc. Orthodiagram, area 127%.		F	52	?	?	O.D. Nervehead edematous and congested; rings absent; arteries reduced in caliber, copper wire appearance and irregular; arteriovenous compression; retina edematous; grayish-white patch in retina; several hemorrhages. O.S. Endarteritis; veins engorged, tortuous and irregular; macula edematous; hemorrhages; otherwise very similar to O.D.
16. A.M. 3/29/18. Diagnosis: Chronic nephritis; hypertension. Urine: Moderate amount of albumin; many R.B.C. and few W.B.C.; granular and hyalin casts. Blood: B.P. 190 to 255; Hgb. 67 to 73%; R.B.C. 4,120,000; W.B.C. 6,700 to 11,000; blood urea 0.042 to 0.060 gms. per 100 cc.		F	34	5/4	5/15	O.D. Disc blurred and swollen about 2 D.; upper nasal quadrant about 3 D.; vessels reduced in caliber and tortuous; some patches of old exudate from old hemorrhages about disc; arteries irregular; retina edematous with scattered areas of exudate; exudate in macula; hemorrhage below macula. O.S. Disc swollen 2 D.; many flame shaped hemorrhages throughout fundus; similar to O.D.
17. L.B. 5/8/18. Diagnosis: Chronic nephritis; hypertension. Urine: Moderate amount of albumin; many hyalin and granular casts; few W.B.C.; phenolphthalein 33% in 2 hours. Blood: B.P. 140 to 240; blood urea 0.057 to 0.066 gms. per 100 cc.; blood sugar 0.14 gms. per 100 cc.		M	53	?	C.F. at 20 in.	O.D. Small hemorrhage at border of disc; marked arteriovenous compression; arteries and veins tortuous and irregular; endarteritis; arteries have silver wire appearance; arteries reduced in caliber; several hemorrhages present; corkscrew vessels. O.S. Rings obscured; vessels almost completely occluded; very tortuous; exudate over disc; retina edematous; vessels followed by whitish streaks of exudate.

18.	A.B. 5/21/18. Diagnosis: Chronic parenchymatous nephritis. Urine: Small amount of albumin; few hyalin and granular casts; phenolphthalein 27 to 30% in 2 hrs. Blood: B.P. not recorded; Hgb. 75%; R.B.C. 4,450,000, W.B.C. 8,116; blood urea 0.035 to 0.180 gms. per 100 cc.	M	53	5/5	5/5	O.D. Disc slightly pale; slight endarteritis; slight edema of retina. O.S. Arteries irregular in caliber; slight retinal edema; essentially same as O.D.
19.	F.H. 6/7/18. Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; many granular casts; phenolphthalein 30% to 40% in 2 hours. Blood: B.P. 150; Hgb. 60%; R.B.C. 3,780,000, W.B.C. 10,000; blood urea 0.021 to 0.036 gms. per 100 cc.	F	25	5/12	5/4-2	O.D. Rings blurred; disc hyperemic and edematous; veins engorged; perivascularitis; arteries reduced in caliber, tortuous and irregular; arteriovenous compression; retina markedly edematous throughout; macula granular. O.S. Same as O.D.
20.	I.D. 6/7/18. Diagnosis: Chronic nephritis. Urine: Small amount of albumin; few granular casts; phenolphthalein 35% in 2 hours. Blood: B.P. 134 to 172; Hgb. 85%; R.B.C. 4,976,000, W.B.C. 7,200; blood urea 0.030 to 0.048 gms. per 100 cc.	M	44	5/5+	5/5+	O.D. Disc slightly edematous; retina slightly edematous; macula granular. O.S. Same as O.D.
21.	C.G. 7/18/18. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; many granular casts; few R.B.C. and few W.B.C.; phenolphthalein 42 to 60% in 2 hours. Blood: B.P. 150 to 170; R.B.C. 3,800,000, W.B.C. 9,000; blood urea 0.033 to 0.052 gms. per 100 cc.	M	36	5/5-3	5/5	O.D. Rings slightly blurred; veins engorged; arterio-venous compression; endarteritis. O.S. Same as O.D.
22.	J.M. 12/19/18. Diagnosis: Chronic nephritis; hypertension. Urine: Moderate amount of albumin; few granular casts; phenolphthalein 11 to 14% in 2 hours. Blood: B.P. 240; blood urea 0.063 to 0.072 gms. per 100 cc. Orthodiagram, area 127%.	M	51	5/5	5/5+	O.D. Nervehead edematous; swollen 1 D.; arteries contracted; endarteritis; arteriovenous compression, veins engorged; retina edematous; some arteries silver wire in appearance; few small hemorrhages; exudates (hemorrhagic) in retina. O.S. Nervehead hyperemic; edematous, swollen 2 D.; rings blurred; otherwise same as O.D.
23.	J.C. 3/8/19. Diagnosis: Chronic nephritis; arterial hypertension. Urine: No albumin, few granular casts; phenolphthalein 18% in 2 hours. Blood: B.P. 185; Hgb. 77%; R.B.C. 3,240,000, W.B.C. 3,800; blood urea 0.040 to 0.052 gms. per 100 cc.	M	46	5/10	5/6-2	O.D. Disc hyperemic and edematous; rings blurred; some arteries small and silver wire in appearance; perivascularitis; old grayish-white exudates; endarteritis; veins irregular; arteries contracted, silver wire, some obliterated; edema of retina; hemorrhages; hyalin deposits in macula. O.S. Marked arteriovenous compression; otherwise same as O.D.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
24.	E.S. 4/10/19..... Diagnosis: Subacute nephritis. Urine: Small amount of albumin; many granular casts and W.P.C.; phenolphthalein 50% in 2 1/6 hours. Blood: B.P. 150; Hgb. 78%; W.B.C. 6,100; blood urea 0.036 gms. per 100 cc.; blood Wassermann negative.	F	24	L.P.	C.F. at 1 ft.	O.D. Rings obscured; cup edematous; arteries tortuous; veins irregular in caliber; arteriovenous compression; some pigment clumping at disc border; absorbing hemorrhages; hemorrhages; some pigment proliferation; macula granular; perivasculitis. Choroidal changes; pigment proliferation; otherwise same as O.D. O.S.
25.	R.P. 10/30/19..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; few granular casts; few W.B.C. and occasional R.B.C.; phenolphthalein 40 to 53% in 2 hours. Blood: B.P. 120 to 125; Hgb. 80%; W.B.C. 9,500; blood urea 0.0276 gms. per 100 cc.	F	19	5/5	5/4	O.D. Nervehead hyperemic; rings blurred; some endarteritis; slight arteriovenous compression; veins engorged and tortuous; one small yellowish-white spot in macular region. O.S. Perivasculitis; otherwise same as O.D.
26.	E.D. 10/31/19..... Diagnosis: Chronic nephritis; uremia. Urine: Large amount of albumin; many hyalin and granular casts; many R.B.C. and W.B.C.; phenolphthalein 18% in 2 hours. Blood: B.P. 230 to 250; Hgb. 70%; R.B.C. 3,750,000; W.B.C. 15,000; blood urea 0.120 gms. per 100 cc.; blood Wassermann negative. Autopsy findings: Chronic parenchymatous nephritis; secondary contracted kidney; uremia.	M	30	?	?	O.D. Whole fundus pale; veins engorged; arteries contracted and irregular; arteriovenous compression; few scattering yellowish spots in macula; one small hemorrhage; marked radiating striations below macula. O.S. Disc swollen and hyperemic; otherwise same as O.D.
27.	E.N. 11/6/19..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; few W.B.C.; no casts; phenolphthalein 50% in 2 hours. Blood: B.P. 190; Hgb. 85%; R.B.C. 5,140,000; W.B.C. 8,200; blood urea 0.027 gms. per 100 cc. Orthodiagram, area 102%.	F	68	5/60	5/60	O.D. Arteries contracted; endarteritis; arteriovenous compression; arteries irregular; veins tortuous; few yellowish-white changes in macula. O.S. Rings blurred; endarteritis obliterans; obliteration of some vessels; marked arteriovenous compression; veins irregular and tortuous; grayish-white irregular area in macula; retina edematous; choroidal sclerosis; one small hemorrhage; yellowish-white dots scattered throughout fundus, mostly superficial.

28.	A.N. 11/12/19. Diagnosis: Chronic nephritis; hypertension. Urine: Large amount of albumin; no casts; phenolphthalein 18% in 2 hours. Blood: B.P. 245 to 280; Hgb. 85%; W.B.C. 9,450; blood urea 0.070 to 0.072 gms. per 100 cc.	F	38	5/60	5/60	O.D. Nervehead swollen between 3 and 4 D.; veins engorged; old hemorrhagic remnants; numerous fleecy changes, some of radiating arrangement in macula; some look like old hemorrhagic remnants. O.S. Disc swollen about 1 D.; rings blurred; endarteritis; veins engorged and tortuous; retina edematous; old hemorrhagic remnants; numerous white dots in macula; many white changes in retina have glistening appearance, not like degenerated blood, but like hyalin tissue.
29.	C.C. 11/14/19. Diagnosis: Chronic nephritis. Urine: Small amount of albumin; no casts; phenolphthalein 30% in 2 hours. Blood: B.P. 140 to 155 Hgb. 85%; W.B.C. 9,800; blood urea 0.060 gms. per 100 cc.	F	56	1/60	6/4	O.D. Disc edematous; arteries irregular; endarteritis; periarteritis; veins engorged; macula edematous; hemorrhages near disc; large body with brilliant reflex, probably vitreous hemorrhage. O.S. Disc edematous; arteriovenous compression; edema of retina.
30.	E.P. 12/27/19. Diagnosis: Chronic nephritis; diabetes mellitus. Urine: Small amount of albumin; many granular casts; sugar present just one day, and cleared up under treatment; phenolphthalein 70% in 2 hours. Blood: B.P. 208; Hgb. 90%; W.B.C. 7,100; blood urea 0.048 gms. per 100 cc.; blood sugar 0.135 to 0.225%.	F	62	5/30	5/12	O.D. Rings blurred; disc edematous; very fine hemorrhages; veins engorged; all along line of vessels are fibrillary hemorrhages; macula edematous; some yellowish spots in macula. O.S. Rings blurred and edematous; arteries irregular, with some exudate covering vessels above disc; veins engorged; periphlebitis; periarteritis; retina and macula edematous; numerous hemorrhages; yellowish spots in macula.
31.	M.B. 1/19/20. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few hyalin casts; few W.B.C.; phenolphthalein 64% in 2 hours. Blood: B.P. 240; Hgb. 90%; R.B.C. 4,900,000, W.B.C. 7,000; blood urea 0.020 gms. per 100 cc.	F	45	5/60	5/20-1	O.D. Nervehead hyperemic and edematous; rings blurred; endarteritis; arteries contracted; veins tortuous; marked arteriovenous compression; edema of retina; early thrombosis temporal vein with hemorrhagic area along artery; veins engorged; old and new hemorrhages; small white plaques around disc and macula. O.S. Nervehead hyperemic and edematous; endarteritis; arteries contracted and almost obliterated in places; white plaque along nasal vein and temporal artery; marked arteriovenous compression; edema of retina; macular region granular.

Medical Findings and Diagnosis				Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
32.	E.H. 2/24/20. Diagnosis: Chronic nephritis; diabetes mellitus. Urine: Large amount of albumin; sugar present only one day and cleared up under treatment; few granular and hyalin casts; phenolphthalein 13% in 2 hrs. Blood: B.P. 165; blood urea 0.035 gms. per 100 cc.	F	57	5/20	5/30	O.D. Disc edematous; rings blurred; endarteritis; veins irregular and tortuous; arteries irregular; retina edematous; fine hemorrhages in macula; not large but round, not flame shaped; groups of spots due to old hemorrhages; flame shaped hemorrhages near macula. O.S. Hemorrhages; similar to O.D.; no typical star formation in macula.		
33.	A.G. 3/1/20. Diagnosis: Chronic nephritis; hypertension; hypertrophy of heart. Urine: Moderate amount of albumin; few hyalin and granular casts; phenolphthalein 37% in 2 hours. Blood: B.P. 210; blood urea 0.056 gms. per 100 cc.; spinal fluid negative. Orthodiagram, area 136%.	F	51	5/10	L.P.	O.D. Rings blurred; endarteritis; arteriovenous compression; superior temporal artery almost completely constricted; arteries very irregular in caliber; retina edematous; macular veins tortuous. O.S. Disc edematous; silver wire arteries; perivascular exudates; periarteritis; retina edematous; endarteritis.		
34.	M.S. 3/8/20. Diagnosis: Chronic nephritis; hypertension. Urine: Moderate amount of albumin; few hyalin and granular casts; many W.B.C.; phenolphthalein 11% to 25% in 2 hours. Blood: B.P. 230 to 250; Hgb. 90%; W.B.C. 8,300, blood urea 0.032 to 0.051 gms. per 100 cc. Patient died—cause of death was hemorrhage into right side of pons, involving III, IV and V nuclei.	F	46	5/10	5/12+	O.D. Nervehead hyperemic and edematous; arteries contracted; veins engorged; small atrophic area above disc; arteriovenous compression; edema of retina; hemorrhage; several white dots in retina. O.S. Nervehead edematous; rings blurred; endarteritis; periarteritis; macula edematous; veins of macula tortuous.		
35.	A.McM. 3/25/20. Diagnosis: Chronic nephritis; hypertension. Urine: Large amount of albumin; many granular casts. Blood: B.P. 240 to 274; blood urea 0.055 to 0.065 gms. per 100 cc.; blood Wassermann and spinal fluid Wassermann negative.	F	36	5/6	C.F.	O.D. Nervehead swollen about 3 D.; edematous and hyperemic; flame shaped hemorrhages; old exudates; arteries contracted; arteriovenous compression marked; one artery entirely obliterated; endarteritis; edema of retina; macula edematous with radiating striations and hemorrhages; silver wire arteries; different shaped hemorrhages; periarteritis; engorged veins; otherwise similar to O.D. including hemorrhages.		
36.	O.M. 4/2/20. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; no casts; phenolphthalein 12 to 38% in 2 hours. Blood: B.P. 160 to 165; Hgb. 75%; R.B.C. 3,440,000; W.B.C. 5,100; blood urea 0.036 to 0.051 gms. per 100 cc.	M	26	5/4-2	5/4-2	O.D. Disc edematous; choroidal change near disc and throughout fundus; looks like minute aggregations of pigment with hyalin change; small hemorrhage near disc. O.S. Disc edematous; rings blurred; arteriovenous compression; arteries contracted; choroidal change same as in O.D.; several hemorrhages.		

37.	C.C. 4/14/20. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; very many granular casts and W.B.C.; few hyaline casts; phenolphthalein only trace in 2 hours. Blood: B.P. 150 to 218; Hgb. 70%; R.B.C. 3,300,000; W.B.C. 6,900; blood urea 0.055 to 0.078 gms. per 100 cc. Orthodiagram, area 176%. Patient died 4/18/20.	M	30	1/60	C.F. at 1 ft.	O.D. Nervehead edematous, swollen 3 D.; rings obliterated; many hemorrhages; old exudates; arteries contracted—silver wire; veins tortuous; edema of retina. whole macula has whitish striations. O.S. Rings obliterated; nervehead swollen 2 D.; arteries contracted; obliterated in places; endarteritis; perivasculitis; hemorrhages; old exudates; veins contracted; macular star radiating out in all directions; edema of retina.
38.	T.M. 4/14/20. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few hyalin and many granular casts; many W.B.C.; phenolphthalein 60 to 65% in 2 hours. Blood: B.P. 105 to 125; Hgb. 100%; W.B.C. 6,700; blood urea 0.019 to 0.033 gms. per 100 cc.	M	37	5/5	5/5	O.D. Rings blurred; nervehead edematous and hyperemic; endarteritis; coppery vessels; arteriovenous compression; silver wire vessels; edema of retina. O.S. Nervehead edematous and hyperemic; perivasculitis; arteriovenous compression; periphlebitis; edema of retina.
39.	L.A. 4/16/20. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; large amount of granular and hyaline casts, in 2 hours. Blood: B.P. 125 to 185; Hgb. 65 to 90%; R.B.C. 3,600,000; W.B.C. 9,900 to 16,500; blood urea 0.012 to 0.066 gms. per 100 cc.	F	13	5/5-2	5/5-1	O.D. Rings blurred; nervehead edematous; edema of retina; macula edematous. O.S. Same as O.D.
40.	H.M. 4/16/20. Diagnosis: Chronic nephritis. Urine: Small amount of albumin; few W.B.C.; phenolphthalein 62% in 2 hours. Blood: B.P. 123; Hgb. 80%; R.B.C. 3,330,000; W.B.C. 8,000; blood urea 0.036 to 0.040 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 105%.	F	28	5/4-3	5/4-1	O.D. Slight edema of nervehead; arteries slightly contracted; endarteritis. O.S. Slight edema of retina; arteries tortuous.
41.	C.B. 5/4/20. Diagnosis: Chronic nephritis. Urine: Large amounts of albumin; very many hyaline granular casts; few R.B.C. and W.B.C.; phenolphthalein 25 to 51% in 2 hours. Blood: B.P. 132 to 170; Hgb. 73 to 90%; R.B.C. 4,100,000; W.B.C. 6,500 to 13,500; blood urea 0.012 to 0.082 gms. per 100 cc.	M	21	5/6-1	5/6-1	O.D. Disc edematous; retina edematous; macula edematous; endarteritis; periarthritis; several areas of choroidal disturbance, throughout whole periphery of fundus. O.S. Disc and retina edematous; macula edematous; endarteritis; periarthritis; choroidal changes as in O.D.

Medical Findings and Diagnosis	Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
<p>42. J.S. 5/19/20. Diagnosis: Chronic nephritis; hypertension. Urine: No albumin; few hyalin casts; few W.B.C.; phenolphthalein 35% in 2 hours. Blood: B.P. 190 to 205; Hgb. 100%; W.B.C. 8,650; blood urea 0.016 to 0.028 gms. per 100 cc.</p> <p>Returns 6/1/20.</p> <p>Urine: (not reported); phenolphthalein 75% in 2 hours. Blood: (not reported); B.P. 170 to 200; blood urea 0.019 to 0.030 gms. per 100 cc.</p>	M	58	1/60	5/15	<p>O.D. Nervehead edematous; rings blurred; arteries markedly contracted; endarteritis; arteriovenous compression; veins engorged; grayish-white areas scattered throughout fundus; many hemorrhages; arteries silver wire; edema of retina; macula pigmented and granular; atrophic changes in macula; coriaceous macular vessels; pigmentary layer of retina atrophic.</p> <p>O.S. Nervehead hyperemic and edematous; obliteration of some of arteries; otherwise same as O.D.</p> <p>O.D. Cup almost obliterated; hemorrhages absorbing; marked endarteritis; some evidence of fresh hemorrhages; nervehead more edematous.</p> <p>O.S. Rings blurred; increased edema of nerve head; recent hemorrhages.</p>
<p>43. M.T. 5/21/20. Diagnosis: Chronic nephritis; hypertension. Urine: Large amount of albumin; few granular casts; many W.B.C.; phenolphthalein 20% in 2 hours. Blood: B.P. 242 to 260; Hgb. 100%; R.B.C. 4,400,000; W.B.C. 9,800; blood urea 0.021 to 0.060 gms. per 100 cc.</p> <p>Returns 6/8/20.</p> <p>Urine: Not reported. Blood: B.P. 225; blood urea 0.045 gms. per 100 cc.</p>	F	49	5/12	5/10	<p>O.D. Veins engorged; endarteritis; arteries contracted, irregular and coppery; arteriovenous compression; retinal edematous; yellowish spot in macula; several hemorrhages, old and recent; numerous exudates, probably due to old hemorrhages.</p> <p>O.S. Disc hyperemic and edematous; retina edematous; veins engorged; hemorrhages; perarteritis; arteriovenous compression; coppery arteries; dull pinkish spot in macula; macula edematous; old and new hemorrhages; endarteritis; obliteration of some of vessels.</p> <p>O.D. Disc blurred; retina hyperemic; edema of disc and retina; macula edematous; radiating spoke like lines; absorbing hemorrhages; fresh ecchymoses, endarteritis; arteries pale yellow in appearance, irregular and contracted.</p> <p>O.S. Macula edematous; radiating spoke like lines extending from macula; choroidal change in macula; arteriovenous compression; edema of retina; old and new hemorrhages; perarteritis; disc swollen 1 D.</p> <p>O.D. Retina edematous; endarteritis; veins engorged; several radiating striations from macula; exudates; hemorrhages.</p> <p>O.S. Rings almost completely veiled; hemorrhages; arteriovenous compression; endarteritis; retina edematous; radiations from macula; macula edematous; deep choroidal changes near macula.</p>
<p>Returns 6/21/20.</p> <p>Urine: (not reported); phenolphthalein 20% in 2 hours. Blood: B.P. 210 to 245; blood urea 0.040 to 0.060 gms. per 100 cc.</p>					

44. M.B. 5/21/20. Diagnosis: Chronic nephritis; hypertension; arterio-sclerosis. Urine: Large amount of albumin; few hyalin and granular casts; phenolphthalein 22% in 2 hours. Blood: B.P. 230 to 242; Hgb. 90%; R.B.C. 5,000,000, W.B.C. 8,500; blood urea 0.052 gms. per 100 cc.	F	69	5/30	5/23	O.D. Disc edematous and hyperemic; endarteritis; hemorrhages; arteriovenous compression; some old white spots of exudate; hemorrhages both round and flame shaped; veins irregular, sausage shaped; retina edematous; aneurysmal dilation along vessel walls; edema of macula. O.S. Disc swollen 1 to 2 D.; rings blurred; retina edematous; many flame shaped hemorrhages; patches of exudate, from old hemorrhages; veins irregular and engorged; endarteritis; arteriovenous compression; arteries contracted; numerous white patches in macula.
45. W.O. 6/8/20. Diagnosis: Chronic nephritis; hypertension. Urine: Large amount of albumin; many granular and hyalin casts; few W.B.C.; phenolphthalein 34% in 2 hours. Blood: B.P. 190; Hgb. 90%; R.B.C. 5,160,000, W.B.C. 14,300; blood urea 0.030 gms. per 100 cc. Patient died of pneumonia 6/17/20. Autopsy: Chronic parenchymatous nephritis.	M	48	5/5	5/5	O.D. Rings blurred; disc edematous; arteries small and irregular; periarthritis; arteriovenous compression; retina edematous; slight exudate; macula edematous. O.S. Same as O.D.
46. F.O. 6/8/20. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; no casts; phenolphthalein 65 to 72% in 2 hours. Blood: B.P. 95 to 130; Hgb. 90%; R.B.C. 4,000,000, W.B.C. 9,400; blood urea 0.041 to 0.062 gms. per 100 cc. Orthodiagram, area 139%.	M	38	5/4	5/4	O.D. Disc hyperemic and margins blurred; veins engorged; arteriovenous compression. O.S. Same as O.D.
47. R.S. 6/12/20. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few hyalin and granular casts; few R.B.C. and W.B.C.; phenolphthalein 46% in 2 hours. Blood: B.P. 155 to 180; Hgb. 100%; R.B.C. 4,680,000, W.B.C. 7,500; blood urea 0.027 to 0.127 gms. per 100 cc. (Patient's urea increased following ingestion of high meat and salt diet.) Orthodiagram, area 109%.	M	49	5/7.5	5/12.1	O.D. Disc edematous and congested; rings blurred; retina edematous; arteries contracted and irregular; endarteritis; periarthritis; arteriovenous compression; one small round exudate. O.S. Disc swollen 1 D. Disc and retina edematous; rings blurred; arteries irregular; endarteritis; arteriovenous compression; small flame shaped hemorrhage, surrounded by exudate; macular veins are tortuous.
Returns 9/29/20. Urine: Moderate amount of albumin; few hyalin casts; few R.B.C. and W.B.C. phenolphthalein 17 to 23% in 2 hours. Blood: B.P. 170 to 200; Hgb. 100%; R.B.C. 5,120,000, W.B.C. 7,100; blood urea 0.024 to 0.058 gms. per 100 cc. Orthodiagram, area 102%.			5/10	5/12	O.D. Disc swollen; macula edematous; beginning radiations in macula; exudate has disappeared. O.S. Disc blurred; numerous yellowish-white dots in macula; small hemorrhage; arteries irregular.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
48.	F.P. 6/14/20..... Diagnosis: Chronic nephritis; hypertension. Urine: Large amount of albumin; few granular casts; phenolphthalein 10 to 34% in 2 hours. R.B.C. Blood: B.P. 195 to 280; Hgb. 54 to 80%; R.B.C. 3,000,000 to 3,870,000, W.B.C. 7,400 to 10,700; blood urea 0.030 to 0.079 gms. per 100 cc.; blood Wassermann negative. Patient died 8/9/20—no autopsy.	M	46	?	?	O.D. Disc edematous; macular vessels tortuous; many new and old hemorrhages; retina edematous; several exudative hemorrhages. O.S. Rings blurred; crescent shaped choroidal change; endarteritis; periarteritis; macular vessels tortuous.
49.	H.T. 6/25/20..... Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few hyalin and granular casts; few W.B.C.; phenolphthalein 25 to 46% in 2 hours. Blood: B.P. 112 to 125; Hgb. 100%; W.B.C. 10,000; blood urea 0.030 to 0.045 gms. per 100 cc. Orthodiagram, area 141%.	M	40	5/7.5-1	5/7.5	O.D. Periarteritis; endarteritis; retina edematous; hyalin dots in macula. O.S. Rings blurred; periarteritis; endarteritis; arterio-venous compression; edema of retina and disc; macula edematous.
50.	C.S. 8/21/20..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; few granular casts; few R.B.C. and W.B.C.; phenolphthalein—no elimination in 5 hours. Blood: B.P. 110; Hgb. 58%; R.B.C. 2,950,000, W.B.C. 17,200; blood urea 0.117 gms. per 100 cc.; blood Wassermann +++.	M	37	5/5-2	5/5-1	O.D. Rings blurred; endarteritis; retina and macula edematous. O.S. One or two dots in macula; otherwise same as O.D.
51.	R.M. 8/23/20..... Diagnosis: Chronic nephritis. Urine: No albumin; few W.B.C.; no casts; phenolphthalein 20% in 2 hours. Blood: B.P. 190 to 200; Hgb. 100%; R.B.C. 4,970,000, W.B.C. 7,600; blood urea 0.024 to 0.033 gms. per 100 cc.; blood sugar 0.12%. Returns 9/14/20..... Blood: B.P. 140 to 195; blood urea 0.028 gms. per 100 cc.	M	61	5/7.5-2	5/5-2	O.D. Disc edematous; slightly pulsating arteries; endarteritis; arteriovenous compression; veins engorged; macula granular and edematous; retina edematous. O.S. Rings blurred; arteries reduced; veins engorged; grayish area; choroidal disturbance; bright spot in macula; numerous fine yellow spots in macula. O.D. Disc margins almost completely obscured; disc edematous and hyperemic; veins engorged; retina hyperemic and edematous; macula edematous. O.S. Disc hyperemic and edematous; veins engorged; rings obscured.

52.	I.D. 9/29/20..... Diagnosis: Chronic nephritis. Urine: Trace of albumin; few R.B.C. casts; phenolphthalein 31 to 68% in 2 hours. Blood: B.P. 115 to 135; Hgb. 90%; R.B.C. 4,890,000; W.B.C. 6,600; blood urea 0.012 to 0.054 gms. per 100 cc. Orthodiagram, area 141%.	M	29	5/10-1	5/7.5-1	O.D. Disc blurred and swollen about 2 D.; veins engorged; endarteritis; disc and retina hyperemic and edematous; macular veins corkscrew like. O.S. Disc swollen 2 D.; veins engorged; endarteritis; macula edematous; corkscrew macular veins.
	Returns 10/26/21.....					O.D. Disc blurred and edematous; endarteritis; arteries contracted; veins engorged; disc and retina both hyperemic and edematous; macula hyperemic and edematous; disc swollen 2 D. O.S. Disc swollen 1½ to 2 D.; same as O.D.
53.	C.B. 11/5/20..... Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few granular and hyalin casts; few W.B.C.; phenolphthalein 47 to 48% in 2 hours. Blood: B.P. 135; Hgb. 95%; R.B.C. 5,340,000; W.B.C. 10,400 to 12,700; blood urea 0.030 to 0.033 gms. per 100 cc. Orthodiagram, area 142%.	M	35	5/6	5/5	O.D. Normal fundus. O.S. Normal fundus.
54.	H.O. 11/13/20..... Diagnosis: Chronic nephritis. Urine: Large amount of albumin; very many hyalin and granular casts; many W.B.C.; no R.B.C.; phenolphthalein 31% in 2 hours. Blood: B.P. 165 to 220; Hgb. 90%; R.B.C. 5,390,000; W.B.C. 7,500; blood urea 0.030 to 0.044 gms. per 100 cc.	M	28	5/4	5/5	O.D. Disc blurred; hyperemic and edematous; retina hyperemic and edematous; endarteritis; perivasculitis; periaertitis; silverwire arteries; veins engorged; old and recent hemorrhages; arteriovenous compression; macula edematous; inflammatory products in macula, giving grayish-white patches. Typical star shaped radiations from macula; otherwise same as O.D. O.S. Typical star shaped radiations from macula; otherwise same as O.D.
55.	F.G. 12/13/20..... Diagnosis: Chronic nephritis. Urine: Small amount of albumin; occasional hyalin cast; few W.B.C.; phenolphthalein 55% in 2 hours. Blood: B.P. 170 to 185; Hgb. 85%; R.B.C. 4,250,000; W.B.C. 6,700; blood urea 0.040 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 131%.	F	32	5/60	5/60	O.D. Some blurring of disc; arteries small; veins small; some choroidal atrophy. O.S. Rings blurred; some localized choroidal disturbance; veins irregular and swollen; mottling of macular region.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
56.	F.R. 12/18/20..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; occasional hyalin cast; few R.B.C. and W.B.C.; phenolsulphathalein 62% in 2 hours. Blood: B.P. 230 to 240; Hgb. 95%; R.B.C. 5,040,000; W.B.C. 9,600; blood urea 0.040 gms. per 100 cc.; blood sugar 0.16%; blood Wassermann negative; spinal fluid Wassermann negative. Orthodiagram, area 140%.	M	35	5/10	5/60	O.D. Disc swollen about 3 D.; rings completely obscured; marked capillary dilatations, suggesting minute hemorrhages; exudates; veins engorged; perivascularitis; arteries contracted; arteriovenous compression; radially mottled striations at border of macula; hyperemia and edema of whole fundus; many exudates, remnants of old hemorrhages. O.S. Disc swollen about 3 D.; endarteritis; periarteritis; new and old hemorrhages; distinct radiating lines in macula having fleecy appearance.
57.	M.I. 1/14/21..... Diagnosis: Chronic nephritis; hypertension. Urine: Moderate amount of albumin; few casts; many W.B.C.; few R.B.C. phenolsulphathalein 66% in 2 hours. Blood: B.P. 150 to 195; Hgb. 88%; R.B.C. 4,770,000; W.B.C. 9,800 to 10,200; blood urea 0.038 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 108%.	F	28	5/10	5/10+3	O.D. Rings blurred; old inflammatory exudate; arteries small and irregular in caliber; endarteritis; veins engorged; arteriovenous compression; numerous small, yellowish spots near disc—several groups also near macula; macula edematous. O.S. Disc swollen about 1 diopter; small hemorrhage; arteries contracted; arteriovenous compression; groups of white dots near disc and in macula.
58.	I.B. 1/19/21..... Diagnosis: Chronic nephritis; arteriosclerosis; chronic myocarditis. Urine: Large amounts of albumin; many granular and hyalin casts; many W.B.C.; phenolsulphathalein 51% in 2 hours. Blood: B.P. 170 to 175; Hgb. 70%; R.B.C. 3,915,000; W.B.C. 8,600; blood urea 0.0396 gms. per 100 cc.; blood Wassermann negative.	M	70	5/5-2	5/15	O.D. Arteriovenous compression; atrophic tasselation of choroid. O.S. Veins tortuous; arteries irregular in caliber; edema of choroid.
59.	H.V.B. 2/2/21..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; few granular and hyalin casts; few W.B.C.; phenolsulphathalein 65% in 2 hours. Blood: B.P. 140; Hgb. 65 to 97%; R.B.C. 3,780,000 to 6,070,000; W.B.C. 9,100 to 10,100; blood urea 0.021 to 0.042 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 113%.	M	27	5/4	5/4	O.D. Perivasculitis; arteries small; endarteritis; periphlebitis; disc and retina hyperemic; slight edema. O.S. Slight edema.
Returns 10/26/21.....						
60.	Blood: B.P. 125; Hgb. 85 to 92%; R.B.C. 4,280,000 to 5,110,000; W.B.C. 9,900 to 10,000; blood urea 0.048 to 0.064 gms. per 100 cc. Orthodiagram, area 124%.			5/4	5/4	O.D. Same as before. O.S. Same as before.

50.	C.C. 2/9/21..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; few hyalin and granular casts; 15 to 49% in 2 hours. Blood: B.P. 100 to 180; Hgb. 95%; R.B.C. 4,460,000; W.B.C. 7,400; blood urea 0.0348 gms. per 100 cc.; blood Wassermann negative; spinal fluid Wassermann negative, area 96%. Orthodiagram, area 96%.	M	45	5/12	5/10	O.D. Rings blurred; arteries reduced in caliber; macula edematous; retina edematous; slight loss of transparency. O.S. Disc hyperemic; rings blurred; smaller veins tortuous; retina edematous.
51.	C.H. 3/24/21..... Diagnosis: Chronic nephritis. Urine: Large amount of albumin; many hyalin and granular casts; 15 to 49% in 2 hours. Blood: B.P. 118 to 150; Hgb. 50 to 80%; R.B.C. 2,340,000 to 4,180,000; W.B.C. 6,600 to 11,500; blood urea 0.016 to 0.034 gms. per 100 cc.; blood Wassermann negative, area 159%. Orthodiagram, area 159%.	M	35	5/20+2	5/20+2	O.D. Disc blurred; arteries tortuous and caliber reduced; loss of transparency throughout retina. O.S. Disc blurred; arteries tortuous; endarteritis; round glistening, yellowish spot down from disc; macula granular, numerous yellowish and hyalin like spots in macula. Retina shows loss of transparency throughout.
52.	C.J. 3/24/21..... Diagnosis: Chronic nephritis; hypertension; arteriosclerosis. Urine: Moderate amount of albumin; few hyalin and granular casts; phenolphthalein 24% in 2 hours. Blood: B.P. 170 to 220; Hgb. 82%; R.B.C. 3,000,000; W.B.C. 13,100; blood urea 0.036 gms. per 100 cc. Orthodiagram, area 171%.	M	60	5/50	C.F. at 1 ft.	O.D. Rings blurred; arteries small and irregular; arterio-venous compression; vessels above disc are obliterated; many hemorrhages; endarteritis; yellowish spots in retina, probably old hemorrhages; edema of macula. O.S. Rings blurred; hemorrhages, new and old; running out from macula is series of confluent macular exudates; irregular macular star, bright yellow in color.
53.	O.F. 4/13/21..... Diagnosis: Chronic nephritis. Urine: Small amount of albumin; few granular casts, R.B.C. and W.B.C. Blood: B.P. 170; Hgb. 90%; R.B.C. 5,340,000; W.B.C. 6,100; blood urea 0.030 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, 97%.	F	50	5/7.5	5/6	O.D. Disc hyperemic; arteries reduced in caliber and tortuous. O.S. Few yellowish spots in macula; otherwise same as O.D.

Medical Findings and Diagnosis			Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
54.	M.M.K. 4/18/21. Diagnosis: Chronic nephritis; hypertension. Urine: No albumin; few hyalin casts; few W.B.C.; phenolphthalein 51% in 2 hours. Blood: B.P. 170 to 205; Hgb. 90%; R.B.C. 5,210,000; W.B.C. 6,600; blood urea 0.031 to 0.054 gms. per 100 cc. Orthodiagram, area 120%.	F	52	5/5+3	5/4-1	O.D. Rings blurred; disc edematous; periarthritis; endarteritis; arteries irregular; arteriovenous compression; retina edematous, hyperemic and congested; disseminated choroidal change below disc; choroid granular with pigment spots; many fine yellowish spots scattered through macula. O.S. Rings blurred; disc and retina edematous, hyperemic and congested; arteriovenous compression; arteries irregular; endarteritis; periarthritis; yellowish spots in macula.	
55.	M.C. 5/11/21. Diagnosis: Chronic nephritis; arteriosclerosis. Urine: Small amount of albumin; few hyalin and granular casts; few W.B.C.; phenolphthalein 60% in 2 hours. Blood: B.P. 170 to 175; Hgb. 90%; R.B.C. 4,280,000; W.B.C. 6,600; blood urea 0.041 to 0.096 gms. per 100 cc.	M	67	5/60	5/60	O.D. Arteries contracted; veins irregular; arteriovenous compression; retina edematous; macula hyperemic and edematous. O.S. Disc edematous; retina edematous; endarteritis; arteries contracted; veins irregular; arteriovenous compression; macula edematous.	
56.	L.S. 5/26/21. Diagnosis: Chronic nephritis; hypertension; diabetes mellitus. Urine: Small amount of albumin; few hyalin casts and W.B.C. Blood: B.P. 170; Hgb. 85%; R.B.C. 3,270,000; W.B.C. 6,300 to 12,000; blood urea 0.028 gms. per 100 cc.; blood sugar 0.21%; blood Wassermann negative.	F	67	?	?	O.D. Atrophic choroid temporally to disc; marked pigment disturbance between macula and disc; arteries reduced in caliber; veins engorged; many hemorrhages; some deposits in macular region; nearly complete conus of disc. O.S. Complete conus of disc; marked sclerotic changes all about disc; pigment changes between disc and macula; arteries reduced in caliber; veins engorged; diffuse hemorrhagic areas throughout retina; some exudates in macular area.	
57.	I.H. 6/28/21. Diagnosis: Chronic nephritis. Urine: Small amount of albumin; few hyalin and granular casts; few R.B.C. and W.B.C.; phenolphthalein 33% in 2 hours. Blood: B.P. 185; Hgb. 100%; R.B.C. 5,220,000; W.B.C. 8,100; blood urea 0.029 gms. per 100 cc. Orthodiagram, area 117 to 123%.	M	60	5/4	5/12	O.D. Disc edematous; endarteritis; arteriovenous compression; some vessels very tortuous; retina edematous between disc and macula. O.S. Same as O.D.	
58.	H.C. 7/9/21. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few W.B.C.; many casts; phenolsulphonethalein 66% in 2 hours. Blood: B.P. 110 to 120; Hgb. 100%; R.B.C. 5,180,000; W.B.C. 9,500; blood urea 0.028 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 118%.	M	36	4/60	4/60	O.D. Rings blurred; disc hyperemic and edematous; retina edematous; slight choroidal change near disc; few hyalin spots in macular region; veins engorged. O.S. Same as O.D.	

59.	M.W. 9/8/21. Diagnosis: Chronic nephritis; hypertension; few R. casts; large amount of albumin; few R. casts; R.P. 150 to 220; Hgb. 75%; R.B.C. 3,990-600; W.B.C. 5,500; blood urea 0.018 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 125%.	F	16	5/6-2	5/4-2	O.D. Rings blurred; disc edematous; whole macula is mottled; choroidal disturbance; choroid edematous; macular region has peculiar mottled, irregularly elevated, snowdrift like appearance. O.S. Same as O.D.
70.	F.R. 9/8/22. Diagnosis: Subacute nephritis; hypertension. W.B.C.; Urine: Moderate amount of albumin; few R.B.C.; few R.P. casts; phenolsulphonethalein 65% in 2 hours. Blood: R.P. 198 to 245; Hgb. 85%; R.B.C. 4,250-000; W.B.C. 7,400 to 12,000; blood urea 0.027 to 0.034 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 116%. Died—autopsy diagnosis; generalized arteriosclerosis; subacute parenchymatous degenerative nephritis.	M	15	5/7.5+2	5/60	O.D. Rings blurred; disc swollen about 1 diopter; endarteritis; arteries contracted; veins engorged; retina edematous; hemorrhages; white, radiating dots in macula; exudate. O.S. Innumerable white dots near disc—give creamy appearance; distinct radiating lines of dots in macula; otherwise same as O.D.
71.	L.L. 10/18/21. Diagnosis: Chronic nephritis; hypertension. W.B.C.; Urine: Large amount of albumin; large number of R.B.C.; R.P. 140; Hgb. 90%; R.B.C. 4,300,000, W.B.C. 9,000; blood Wassermann negative. Orthodiagram, area 156%.	F	44	?	?	O.D. Endarteritis; edema of disc and retina. O.S. Rings blurred; veins tortuous; endarteritis; arterio-venous compression.
72.	M.R. 10/20/21. Diagnosis: Chronic nephritis; hypertension. Urine: Large amount of albumin; many granular and hyaline casts; W.B.C. and R.B.C.; phenolsulphonethalein 28% in 2 hours. Blood: R.P. 186 to 200; Hgb. 80%; R.P.C. 4,100-000; W.B.C. 8,200 to 19,600; blood urea 0.024 to 0.074 gms. per 100 cc.; blood Wassermann negative.	F	32	?	?	O.D. Rings blurred; many small, brilliant, white spots between macula and disc, having arrangement like wheel spokes; retina edematous; some variation in caliber of arteries. O.S. Hemorrhages; white, brilliant spots, with no regular arrangement; otherwise same as O.D.

Medical Findings and Diagnosis		Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
73.	W.B. 10/26/21..... Diagnosis: Chronic nephritis. Urine: Small amount of albumin; few W.B.C.; phenolsulphonethalein 60% in 2 hours. Blood: B.P. 100; Hgb. 95%; R.B.C. 4,000,000, W.B.C. 6,200; blood urea 0.022 gms. per 100 cc.; blood Wassermann +++. Orthodiagram, area 104%.	M	50	5/15	5/15	O.D. Pigment layer of eye atrophic; hyalin dot in center of fovea. O.S. Same as O.D.
74.	A.S. 11/6/21..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; small amount of sugar; few W.B.C.; few hyalin casts; phenolsulphonethalein 25% in 2 hours. Blood: B.P. 130; Hgb. 95%; R.B.C. 5,100,000, W.B.C. 5,500; blood urea 0.026 gms. per 100 cc.; blood sugar 0.16% Orthodiagram, area 138%.	M	36	?	?	O.D. Disc blurred; hyperemic; veins engorged; loss of transparency of retina. O.S. Same as O.D.
75.	C.G. 11/7/21..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; few hyalin casts; few W.B.C.; phenolsulphonethalein 70% in 2 hours. Blood: B.P. 142; Hgb. 100%; R.B.C. 4,800,000, W.B.C. 6,200; blood urea 0.050 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 183%.	F	57	?	?	O.D. Vessels in disc are veiled; slight endarteritis; veins tortuous. O.S. Same as O.D.
76.	E.H. 11/8/21..... Diagnosis: Chronic nephritis. Urine: Moderate amount of albumin; few hyalin and granular casts; phenolsulphonethalein 35 to 40% in 2 hours. Blood: B.P. 140; Hgb. 110%; R.B.C. 5,990,000, W.B.C. 9,600 to 18,400; blood urea 0.025 gms. to 100 cc.; blood Wassermann negative. Orthodiagram, area 107%.	M	35	5/5-2	5/5-1	O.D. Disc and retina hyperemic; some veiling of disc border; arteries small and irregular in caliber; edema of disc and retina; whole fundus hyperemic. O.S. Disc swollen and edematous, and hyperemic. Rings almost obscured; arteries irregular and small; veins engorged; retina hyperemic; loss of transparency and edematous; macula edematous; whole fundus hyperemic.
77.	F.B. 11/9/21..... Diagnosis: Chronic nephritis; hypertension. Urine: Large amount of albumin; few W.B.C. and R.B.C.; many granular and hyalin casts; phenolsulphonethalein under 10% in 2 hours. Blood: B.P. 260 to 300; Hgb. 89%; R.B.C. 4,780,000, W.B.C. 10,900; blood urea 0.020 to 0.024 gms. per 100 cc.; blood Wassermann negative.	M	47	?	?	O.D. Disc red and edematous; swollen 2 diopters; margins of disc completely blurred; retina edematous; veins engorged and tortuous; marked arterio-venous congestion; arteries small, contracted and tortuous; hemorrhages; many fine, glistening white points all about macula; exudates. O.S. Same as O.D.

78.	R.A. 11/28/21..... Diagnosis: Chronic nephritis. Urine: Large amount of albumin; many granular casts; many W.B.C.; phenolsulphonethalein 10 to 28% in 2 hours. Blood: B.P. 160 to 198; Hgb. 70%; R.B.C. 3,820,000; W.B.C. 8,200; blood Wassermann negative; nonprotein nitrogen 0.034 to 0.096 gms. per 100 cc.	M	33	5/3-2	5/3-4	O.D. Disc edematous; arteries contracted; macular region darkly granular and studded with minute white dots. O.S. Choroidal disturbance; otherwise same as O.D.
79.	B.G. 12/27/21..... Diagnosis: Chronic nephritis; hypertension; arterio-sclerosis. Urine: Moderate amount of albumin; few casts; few W.B.C.; phenolsulphonethalein 28% in 2 hours. Blood: B.P. 215 to 230; Hgb. 80%; R.B.C. 4,870,000; W.B.C. 9,900; nonprotein nitrogen 0.045 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 200%.	M	50	5/60	5/10-3	O.D. Rings blurred and nearly obscured; disc swollen 1 diopter; arteries small and irregular; veins tortuous; edema of macula; hemorrhages and exudates; edema of retina; peculiar white striations in choroid; periphlebitis. O.S. Arteriovenous compression; some white spots in macula; otherwise same as O.D.
30.	G.V.O. 1/17/22..... Diagnosis: Chronic nephritis; chronic myocarditis. Urine: Moderate amount of albumin; many W.B.C.; many hyalin casts; few granular casts; phenolsulphonethalein shows a diminished output. Blood: B.P. 140; Hgb. 90%; R.B.C. 4,300,000; W.B.C. 8,900; nonprotein nitrogen 0.036 gms. per 100 cc. Orthodiagram, area 210%.	F	42	?	?	O.D. No gross fundus lesion. O.S. No gross fundus lesion.
Returns 2/15/22.....						O.D. Disc edematous; rings blurred; endarteritis; veins engorged and tortuous. O.S. Same as O.D.
81.	I.R. 1/18/22..... Diagnosis: Chronic nephritis. Urine: Large amount of albumin; few W.B.C.; many granular casts; phenolsulphonethalein 55% in 2 hours. Blood: B.P. 110 to 120; Hgb. 100%; R.B.C. 4,860,000; W.B.C. 7,600; nonprotein nitrogen 0.036 to 0.054 gms. per 100 cc. Orthodiagram, area 145%.	M	35	?	?	O.D. Normal fundus. O.S. Disc hyperemic; rings blurred; arteries tortuous.

	Medical Findings and Diagnosis	Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
82.	J.E. 2/18/22..... Diagnosis: Chronic nephritis. Urine: Small amount of albumin; few casts; phenolsulphonphthalein 35% in 2 hours. Blood: B.P. 185 to 190; Hgb. 76%; R.B.C. 4,300,000; W.B.C. 8,500; nonprotein nitrogen 0.024 gms. per 100 cc.; blood Wassermann negative.	M	32	5/60	5/7.5	O.D. Disc blurred and hyperemic; rings blurred; hemorrhages and exudates; edema of disc and retina; arteries small; endarteritis; arteriovenous compression; fan shaped radiating lines from fovea. O.S. Same as O.D.
33.	B.L. 3/14/22..... Diagnosis: Chronic nephritis; hypertension; arteriosclerosis. Urine: Small amount of albumin; many W.B.C.; phenolsulphonphthalein 68% in 2 hours. Blood: B.P. 180; Hgb. 92%; R.B.C. 4,600,000; W.B.C. 5,800; nonprotein nitrogen 0.021 gms. per 100 cc.; blood Wassermann negative.	M	35	5/6-2	5/5-3	O.D. Rings blurred; veins large; arteries irregular; arteriovenous compression; fine white dots in macula; disc, retina and macula edematous. O.S. Same as O.D.
84.	R.F. 4/7/22..... Diagnosis: Chronic nephritis; chronic myocarditis. Urine: Small amount of albumin. Blood: B.P. 140 to 150; Hgb. 58%; R.B.C. 4,940,000; W.B.C. 11,100; nonprotein nitrogen 0.024 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 185%.	F	50	5/60	5/60	O.D. Rings blurred; cup blurred; perivasculitis; endarteritis; veins engorged; macula edematous; many yellowish-white dots near macula. O.S. Same as O.D.
85.	W.F. 4/11/22..... Diagnosis: Chronic nephritis. Urine: No albumin; no casts; phenolsulphonphthalein 60% in 2 hours. Blood: B.P. 200 to 212; Hgb. 90%; R.B.C. 4,100,000; W.B.C. 10,500; nonprotein nitrogen 0.026 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 122%. Returns 5/2/22.....	M	53	5/7.5-2	5/7.5-1	O.D. Disc swollen 1 diopter; rings blurred; arteries coppery, irregular in caliber; perivasculitis; hemorrhages; choroidal changes; exudates; veins engorged; arteriovenous compression; macula edematous; hyalin dots in macula; some retinal dots suggesting radial arrangement near foveola. O.S. Same as O.D.
				5/6-2	5/6-3	O.D. Hemorrhages; radial dotted macular appearance more striking. O.S. Macular change more distinct; radial arrangement definitely present.
86.	J.F. 4/20/22..... Diagnosis: Chronic nephritis; hypertension. Urine: Small amount of albumin; few R.B.C.; few W.B.C.; few hyalin casts; phenolsulphonphthalein 50% in 2 hours. Blood: B.P. 220 to 225; Hgb. 93%; R.B.C. 4,480,000; W.B.C. 7,400; nonprotein nitrogen 0.028 gms. per 100 cc. Orthodiagram, area 124%.	F	60	5/10	5/20	O.D. Disc edematous; endarteritis; arteriovenous compression; veins tortuous and engorged; edema of retina; arteries contracted and tortuous. O.S. Same as O.D.

14, 4/20/22. Diagnosis: Chronic nephritis. Urine: Large amount of albumin; many casts; few R.B.C. and W.B.C.; phenolsulphonethalein 51% in 2 hours. Blood: B.P. 140 to 160; Hgb. 72%; R.B.C. 4,560,000; W.B.C. 5,800; nonprotein nitrogen 0.027 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 105%.	F	47	5/4-3	5/4-2	O.D. O.S.	Rings blurred; disc edematous; arteries small and contracted; veins engorged and tortuous; arteriovenous compression; retina edematous. Choroidal disturbance; few white dots in macula; otherwise same as O.D.
37. C.W. 5/4/22. Diagnosis: Chronic nephritis; hypertension. Urine: Small amount of albumin; few hyalin and granular casts; few W.B.C.; phenolsulphonethalein 33% in 2 hours. Blood: B.P. 218; Hgb. 100%; R.B.C. 4,400,000; W.B.C. 5,400; nonprotein nitrogen 0.030 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 216%.	M	33	5/7-1	5/7-5-1	O.D. O.S.	Rings blurred; disc hyperemic and swollen 1 diopter; endarteritis; arteriovenous compression; veins engorged; arteries irregular and tortuous; perivascularitis; hemorrhages; retina edematous; group of white, glistening spots in macula, having spoke like arrangement. Choroidal disturbance interspersed with fine white dots; otherwise same as O.D.
38. I.K. 5/8/22. Diagnosis: Chronic nephritis. Urine: Small amount of albumin; few hyalin and granular casts; few W.B.C.; phenolsulphonethalein 29% in 2 hours. Blood: B.P. 102 to 150; Hgb. 72%; R.B.C. 4,420,000; W.B.C. 8,100; nonprotein nitrogen 0.029 gms. per 100 cc.; blood Wassermann negative. Orthodiagram, area 136%.	M	60	5/6-2	5/6-3	O.D. O.S.	Veins engorged and tortuous; arteriovenous compression; variations in arterial caliber; disc and retina edematous. Same as O.D.
39. F.W. 5/19/22. Diagnosis: Chronic nephritis. Urine: No albumin; many W.B.C.; few R.B.C.; no casts. Blood: B.P. 100; Hgb. 95%; R.B.C. 4,800,000; W.B.C. 11,800; nonprotein nitrogen 0.030 gms. per 100 cc. Orthodiagram, area 210%.	M	36	5/12-1	5/10+3	O.D. O.S.	Rings blurred; slight perivascularitis; veins full. Endarteritis; arteries small, macula edematous.

TABLE 4.—SERIES D—ACUTE NEPHRITIS

Medical Findings and Diagnosis	Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
1. A.B. 9/21/20. Diagnosis: Acute nephritis. Urine: Small amount of albumin; few R.B.C., W. B.C. and casts; phenolsulphonephthalein 22% to 48% in 2 hours. Blood: B.P. 110 to 140; Hgb. 85%; R.B.C. 5,020, 000; W.B.C. 8,200 to 11,600; blood urea 0.028 to 0.041 gms. per 100 cc.; Wassermann negative.	M	17	5/5-2	5/6-1	O.D. Disc hyperemic; several yellowish changes in ma- cula, pin point and larger; macula edematous; slight endarteritis; periarteritis. O.S. Very small hemorrhage; otherwise same as O.D.

TABLE 5.—SERIES E—RETINAL CONDITIONS SUGGESTING NEPHRITIS

Ocular Findings	Sex	Age	Vision O.D.	Vision O.S.	Ocular Findings
1. M.P. 11/23/17. Diagnosis: Cardiorenal. Urine: No albumin; no casts; few W.B.C.; phenol- sulphonephthalein 45% in 2 hours. Blood: B.P. 148; Hgb. 75%; W.B.C. 8,100; blood urea 0.040 gms. per 100 cc.; Wassermann negative.	F	55	?	?	O.D. Arteries engorged; endarteritis; macula granular. O.S. Arteriovenous compression; slight central retinitis.
2. J.K. 10/23/20. Diagnosis: Chronic nephritis (?). Urine: No albumin; many W.B.C. Blood: B.P. 115; Hgb. 83%; R.B.C. 4,690,000, W. B.C. 9,000; blood Wassermann negative; blood urea 0.090 gms. per 100 cc (?).	F	50	w.c. 5/5-2	w.c. 5/5-1	O.D. Arteries small and tortuous; arteriovenous com- pression; several white spots near fovea, with small pigment spot below; macula coarsely granu- lar; choroid hyperemic; edema of the retina. O.S. Disc edematous; rings blurred; endarteritis; macula edematous.

In table 7, the predominating fundus lesions are so grouped, that the actual number of cases presenting the various lesions may be compared in relation to the principal laboratory findings in the general examination. Graphic chart No. 2 represents the totals in Table 7. These totals are first reduced to percentages as they appear in each series, and the percentages are then represented by the horizontal bars as they would appear in an equal number of cases.

TABLE 6—(SUMMARY OF TABLES 1-5)—IMPORTANT FUNDUS LESIONS AND THE DIFFERENT TYPES OF CONSTITUTIONAL DISEASES GROUPED FOR COMPARISON.

	A.		B.		C.		D.		E.			
	Ocular Totals No.	%	Chronic Interstitial Nephritis No.	%	Hyperten- sion No.	%	Chronic Nephritis No.	%	Acute Nephritis No.	%	Miscel- laneous No.	%
Total cases	136	100	23	16.9	20	14.7	90	66.1	1	0.73	2	1.46
Male	76	55.8	13	56.5	7	35	55	61.1	1	100
Female	60	44.2	10	43.5	13	65	35	38.9	2	100
Average age	45	57.6	..	54.3	..	43.9	17	52.5
1. Edema of disc, macula or retina.....	124	91.1	22	95.6	18	90	82	91.1	1	100	1	50
2. Congestion or slight swelling of disc.....	48	35.3	4	17.4	4	20	39	43.3	1	100
3. Swelling of disc 1 D or more	19	14	1	5	18	20
4. Periarteritis	39	28.8	5	21.7	5	25	28	31.1	1	100
5. Endarteritis	80	59	14	60.9	13	65	51	56.6	1	100	1	50
6. Irregular, tortuous or contracted arteries.....	100	73.5	18	78.2	16	80	64	71.1	2	100
7. Silver wire arteries..	12	8.8	1	4.3	1	5	10	11.1
8. Corkscrew vessels ..	9	6.6	4	17.4	2	10	3	3.3
9. Arteriovenous compression with or without dilatation...	92	67.6	18	78.2	17	85	55	61.1	2	100
10. Irregular, engorged or tortuous veins..	94	69.1	18	78.2	14	70	62	68.8
11. Peripblebitis	5	3.6	1	5	4	4.4
12. Venous thrombosis..	1	0.7	1	1.1
13. Hemorrhages	49	36	7	30.4	3	15	38	42.2	1	100
14. Exudative and other retinal changes	48	35.3	6	26	2	10	40	44.4
15. Retinal detachment..
16. Radiating or star shaped macular changes	16	11.7	1	4.3	15	16.6
17. Other macular changes	50	36.7	7	30.4	7	35	33	36.6	1	100	2	100
18. Choroidal changes..	34	25	7	30.4	4	20	22	24.4	1	50
19. Aneurysmal dilatations or varicosities.....	4	2.9	2	8.6	2	2.2
20. Atropic characteristics	13	9.5	1	4.3	1	5	11	12.2
21. Inflammatory characteristics	85	62.5	12	52.2	10	50	60	66.6	1	100	2	100

Turning to Graphic chart No. 1, the first feature, edema, present in 91 percent of the cases, is more frequent in interstitial nephritis than in hypertension or chronic nephritis. In hypertension, it was slightly less frequent than in chronic nephritis. In the former series, these relations, while not the same, were so similar, that the conclusion that edema in the fundus is probably of vascular origin is well supported.

Congestion or slight swelling of the disc was found in 35 percent of all cases. It was present in 17 percent of inter-

the swelling amounted to at least 1 D., this suggestion is still stronger; for while there were no such cases found in interstitial and only 5 percent in hypertension, 20 percent of the cases of chronic nephritis showed marked swelling of the disc, and nearly all of these cases showed macular lesions. Referring to table 7, it is seen that all but one of these cases showed increased blood pressure, and all but three high blood pressure, an observation that was made in the previous paper.

Periarteritis was present in 21 percent of interstitial nephritis, 25 per cent of hypertension, and 31 percent of parenchymatous nephritis, while endarteritis was seen in 61 percent of interstitial nephritis, 65 percent of hypertension, and 56 percent of parenchymatous. Periarteritis is therefore somewhat more indicative of kidney pathology, while endarteritis would seem to point to hypertension, with interstitial kidney changes as a probable accompaniment.

Changes in the caliber and tortuosity of the arteries were found in 80 percent of hypertension, 78 percent interstitial nephritis, and 71 percent of parenchymatous nephritis. In the previous series, such changes were rather more frequent in the interstitial cases than in hypertension, otherwise the relationship was similar. Their slight preponderance in hypertension and in interstitial over parenchymatous nephritis, would again appear to show that there are factors other than arterial changes present in nephritis.

Silver wire arteries, most frequent in the first series in interstitial nephritis and hypertension, and in parenchymatous nephritis in the second series, are probably a late stage of vascular disease, where all the coats become thickened, the caliber greatly reduced and the size diminished, rather than a process due to the kidney involvement.

Corkscrew like vessels in our cases have almost exclusively been confined to macular veins. In the first series, they were most frequent in parenchymatous nephritis, next frequent in interstitial nephritis, and least in hypertension; on the other hand, in the second series they were most frequently observed in interstitial nephritis, and least frequently in parenchymatous. We are unable to explain this discrepancy, but in view of the larger number of cases in the second series, it would seem that they are an accompaniment of hypertension rather than nephritis *per se*.

Arteriovenous compression, found in 85 percent in hypertension, 78 percent in interstitial, 61 percent in parenchy-

matous nephritis, is probably mainly an evidence of hyperpiesis. In the previous series, such crossings were more frequent in interstitial nephritis than in hypertension, leading to the same conclusion, for all of these cases in the first series showed marked hyperpiesis.

The same remarks apply with nearly equal force in irregular, engorged or tortuous veins, and the high percentage—78 in interstitial nephritis, 70 in hypertension and 68.8 in parenchymatous nephritis—indicate their diagnostic value in high blood pressure.

Periphlebitis was more frequently noted in the first than in the second series, though their relative frequency was about the same, being more often present in both series in parenchymatous nephritis.

Venous thrombosis is probably an accidental feature. In the first series there was one case in the hypertension group; in the second series one in the parenchymatous group.

Hemorrhages, exudates and other retinal changes in the first series appeared to be more characteristic of hyperpiesis, while in the second series both were found much more frequently in parenchymatous nephritis. As in other instances where the findings are more or less at variance, the difference is probably due to the better perspective afforded by the much larger number of cases (90) of parenchymatous nephritis studied, in comparison with 18 cases reported in the first series. The conclusion, therefore, that hemorrhages and exudates are, in part, at least, toxic in origin seems well supported.

Although there were four cases of retinal detachment in the first series, none were observed in the second.

The distribution of radiating macular changes arrests attention in both series. There was only one case in each series in the chronic interstitial group, while 22 per cent of the first and 16.6 percent of the second series of parenchymatous nephritis showed this feature. It, therefore, seems almost certain that the radiating macular changes, so often described as being found in a late stage of interstitial nephritis, are rather a distinctive feature of a late toxic stage of chronic parenchymatous disease.

Other macular changes vary but little in the different groups. They were rather more frequent in the first series in hypertension and interstitial forms, while in the second series, a slightly greater percentage of cases were observed

Table 7B.—Predominating Retinal Lesions, as Associated With the Following Laboratory Findings Relating to the General Disease.

	(B) Hypertension—20 cases.									
	Number cases of each laboratory feature.	Edema, congestion or slight swelling of disc.	Swelling of disc 1 D. or more.	Arterial changes and tortuosities.	Silver wire and corkscrew arteries.	Arteriovenous compression.	Changes or tortuosities in veins.	Hemorrhages.	Exudates and other retinal changes.	Other macular changes.
High blood pressure.....	12	9	1	11	1	11	9	2	..	6
Moderate blood pressure.....	8	8	..	6	2	6	6	2	..	2
Normal or nearly normal blood pressure	0
Slow elimination.....	3	3	..	2	..	3
Normal or fair elimination.....	14	12	1	13	2	11	8	2	..	6
Amount of albumin moderate or large...	1	1	..	1	..	1	1
Amount of albumin, small or absent...	19	16	1	16	3	16	15	4	..	7
Low red blood cell count.....	3	3	..	2	1	3	2	2	..	1
High white blood cell count.....	3	2	..	3	1	2	3	2
Normal blood urea.....	11	11	1	9	2	9	8	2	..	6
Increased blood urea.....	4	3	..	3	1	4	3	1	..	1
Enlarged heart.....	8	8	1	5	1	7	5	2	..	2
Blood Wassermann ± or + + + +	0

Table 7C.—Predominating Retinal Lesions, as Associated With the Following Laboratory Findings Relating to the General Disease.

	(C) Chronic Nephritis—90 cases.									
	Number cases of each laboratory feature.	Edema, congestion or slight swelling of disc.	Swelling of disc 1 D. or more.	Arterial changes and tortuosities.	Silver wire and corkscrew arteries.	Arteriovenous compression.	Changes or tortuosities in veins.	Hemorrhages.	Exudates and other retinal changes.	Other macular changes.
High blood pressure.....	25	25	13	25	6	25	25	25	13	13
Moderate blood pressure.....	21	16	2	19	3	13	10	10	1	8
Normal or nearly normal blood pressure	34	28	1	25	2	15	24	4	3	10
Slow elimination.....	51	40	9	44	9	30	30	26	28	16
Normal or fair elimination.....	32	24	5	24	1	20	25	9	8	12
Amount of albumin, moderate or large...	56	45	13	49	7	32	40	28	28	16
Amount of albumin, small or absent...	35	26	3	28	4	22	23	14	11	16
Low red blood cell count.....	24	21	2	21	2	11	15	11	12	8
High white blood cell count.....	30	27	5	28	1	15	19	15	6	12
Normal blood urea.....	58	46	9	51	6	35	39	23	24	25
Increased blood urea.....	26	22	7	24	5	16	21	18	16	9
Enlarged heart.....	32	25	8	28	5	17	23	12	12	9
Blood Wassermann ± or + + + +	2	1	..	1	2

high blood pressure alone is one of the causes of these features, for they are even more striking in moderate or normal blood pressure, although in parenchymatous cases they are somewhat less numerous.

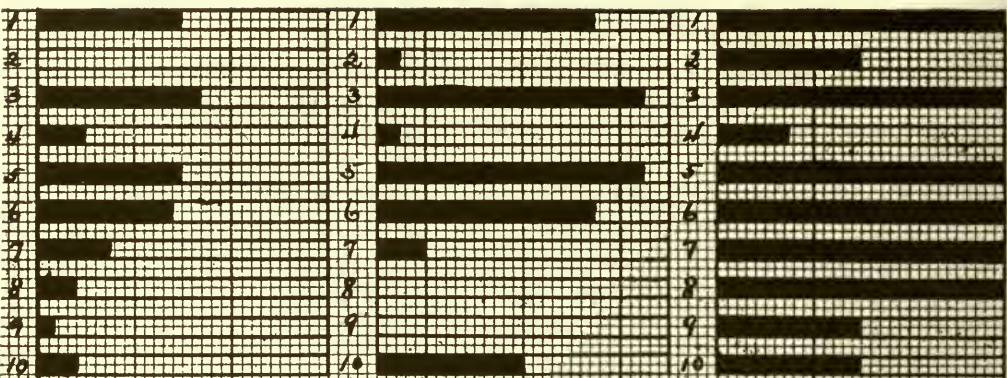
Swelling of the disc 1 D. or more, with exception of one case of hypertension, appears only in the parenchymatous group, and with decreasing frequency as the blood pressure becomes normal. This feature is, therefore, probably dependant upon both increased blood pressure and toxemia, associated with considerable albumin, increased blood urea, and cardiac hypertrophy.

Arterial changes are prominent features in all the graphs, though they also decrease in the parenchymatous group as the blood pressure becomes normal. It was a matter of some surprise that they were found less frequently in this series in the interstitial group, especially in as much as they were most frequent in this group in the former series. When the two groups are combined, they were seen in nearly 75% of the 53 interstitial cases. They are probably an index of the extent of the vascular disease.

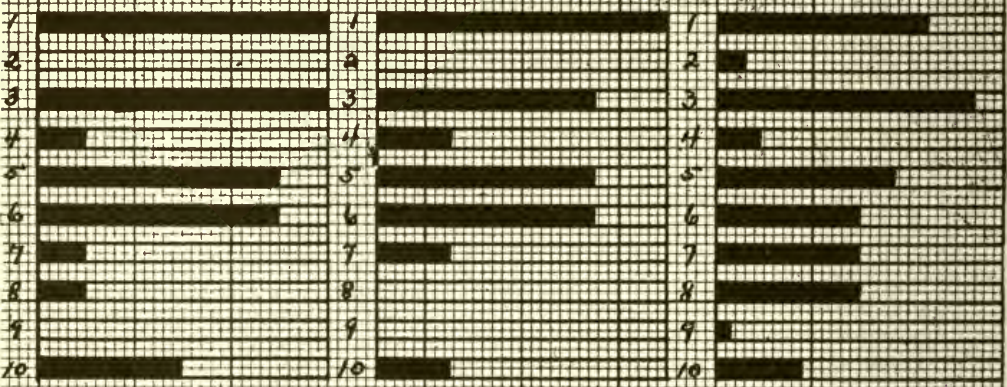
In high blood pressure, silver wire arteries were rather frequent in both forms of nephritis, but most frequent in the parenchymatous group. When the blood pressure was moderate, they were observed more often in hypertension. With normal blood pressure, they were seen only in chronic parenchymatous nephritis. Their distribution in the present or in the former series throws little if any light on their etiology, but taking the two series conjointly, they appear to be a vascular disease pure and simple, and mainly characteristic of advanced cases.

In high blood pressure, the distribution of the feature arteriovenous compression (No. 5) appears to be closely related to arterial changes (No. 3), silver wire arteries (No. 4), and to changes in the veins (No. 6), their frequency being greatest in the parenchymatous group. Their relationship is reversed in moderate blood pressure, and again reversed where the blood pressure is normal. This feature reappears in all the graphs so regularly, rising and falling with increase or decrease of silver wire arteries (No. 4), that it may be regarded as having about the same significance.

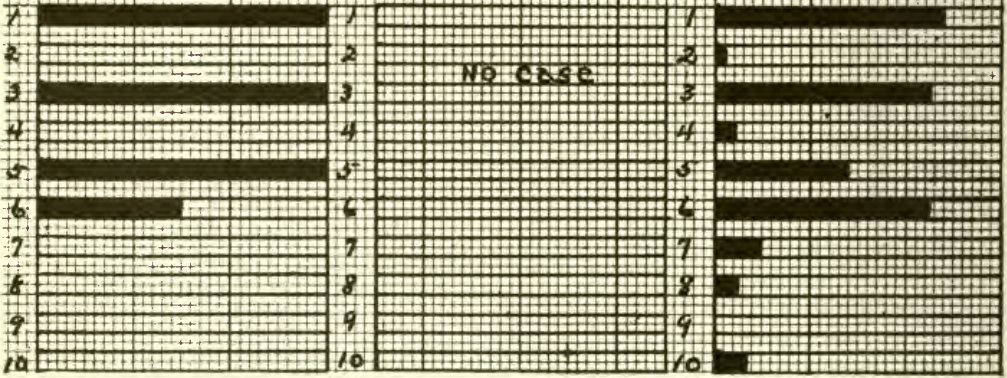
In high or moderate blood pressure, changes or tortuosities in the veins (No. 6) bear a close relationship to the feature just considered (No. 5). In normal blood pressure, they



Cases in which high blood pressure was a prominent feature



Cases in which high blood pressure was not a prominent feature



Cases which showed normal blood pressure.

Chart 2. Section 1. Graphic representation of totals and percentages in Table 7. Each group represents the percentages as they would appear in an equal number of cases, the grouping being based on the laboratory findings in the general examination instead of the diagnosis: 1. Edema, congestion or slight swelling of the disc. 2. Swelling of disc 1 D. or more. 3. Arterial changes or tortuosities. 4. Silver wire and corkscrew arteries. 5. Arteriovenous compression. 6. Changes or tortuosities in veins. 7. Hemorrhages. 8. Exudates and other retinal changes. 9. Radiating macular changes. 10. Other macular changes.

become more prominent in the parenchymatous group, and this prominence continues in about the same relative frequency in nearly all of the graphs, in most of which, notwithstanding the disparity in number, they seem closely related to swelling of the disc 1 D. or more, and, therefore, associated not only with arterial changes, but also with striking regularity with toxemia, increased blood urea, considerable albumin in the urine, and enlargement of the heart.

While hemorrhages and exudates (7 and 8) characterize all the cases of parenchymatous nephritis where the blood pressure was high, and diminish as the pressure becomes moderate, they are present only in this group in normal blood pressure. In the hyperpiesis group, exudates did not appear, although hemorrhages were frequently observed. Their frequency bears a close relation to changes in the veins (No. 6), excepting in normal blood pressure. In as much as in all other graphs, with exception of the two cases of positive Wassermann reaction, they become prominent as the laboratory findings indicate impaired kidney function and cardiac hypertrophy, the conclusion that they are in part at least toxic and perhaps nephritic toxic is clearly suggested. While these relations were somewhat different in the first series, the much smaller number in the parenchymatous group, 18 as compared with 90 in this series, emphasizes the danger of positive conclusions being drawn from a limited number of cases.

Radiating macular changes (No. 9) were not seen where the blood pressure was low, probably because they rarely appear until a relatively late stage of the disease, when the blood has become burdened with toxic products. Their frequency when the white blood cell count is high indicates the possibility of an inflammatory element.

The other macular changes (No. 10) appear to be somewhat associated with increased blood pressure, but they also come out with some prominence where the white blood count is high, in enlargement of the heart, and in the presence of a positive Wassermann.

In slow elimination, swelling of the disc 1 D. or more is present in nearly 20% of the parenchymatous group, and in over 16% of normal or fair elimination. The rapidity of the elimination, however, appears to have very little influence on those features which in our study seem to be vascular in character

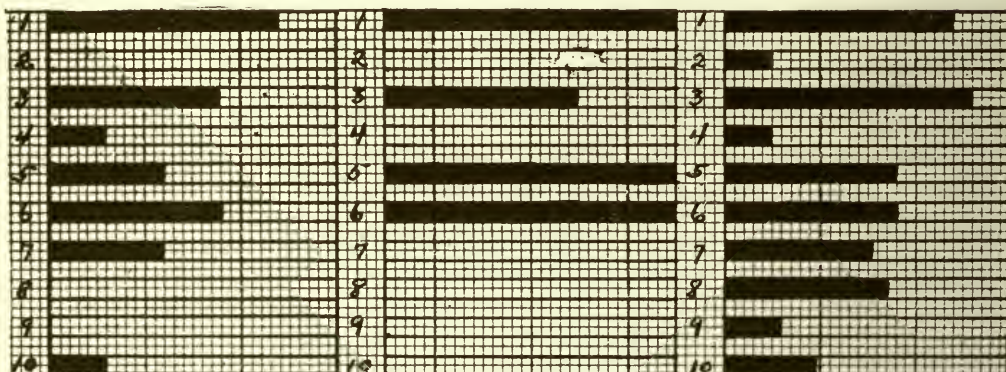
and causation. On the other hand, the features which appear to have a more distinctly toxic relation, hemorrhages (No. 7), exudates (No. 8), and radiating macular changes (No. 9), are more common in slow elimination in the parenchymatous group. Where the elimination is good, radiating macular changes are infrequent. Other macular changes (No. 10) are more prominent in good elimination, suggesting their vascular rather than toxic relation.

When a moderate or large amount of albumin appears in

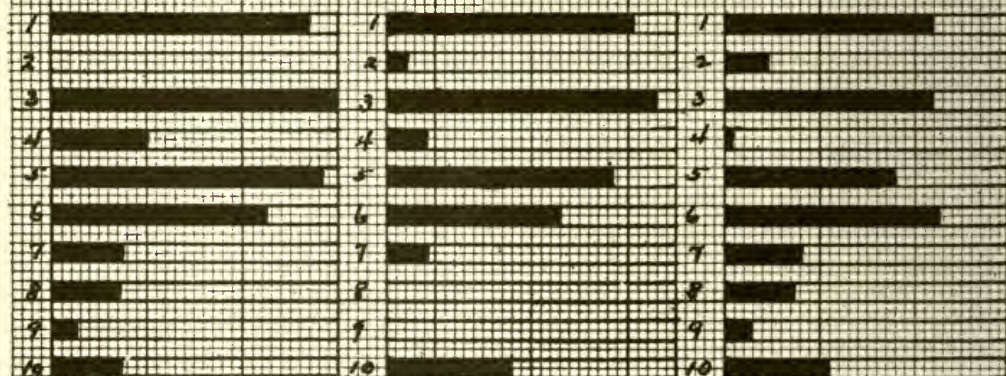
A
Chronic Interstitial Nephritis

B
Hypertension

C
Chronic Nephritis



Cases which showed Slow elimination



Cases which showed normal elimination or only slightly impaired

Chart 2. Section 2. 1. Edema, congestion or slight swelling of the disc. 2. Swelling of disc 1 D. or more. 3. Arterial changes or tortuosities. 4. Silver wire and corkscrew arteries. 5. Arteriovenous compression. 6. Changes or tortuosities in veins. 7. Hemorrhages. 8. Exudates and other retinal changes. 9. Radiating macular changes. 10. Other macular changes.

the urine, in the parenchymatous group, swelling of the disc (No. 2) is much more frequent, changes in the veins (No. 6) are slightly more prominent, hemorrhages (No. 7) more common and exudates (No. 8) still more often present. Radiating macular changes (No. 9) are more frequent when the albumin was small in this series, reversing this feature in the first series, which perhaps shows that the toxic element is not necessarily dependent upon the amount of albumin. In the one case showing this feature in the interstitial group, the

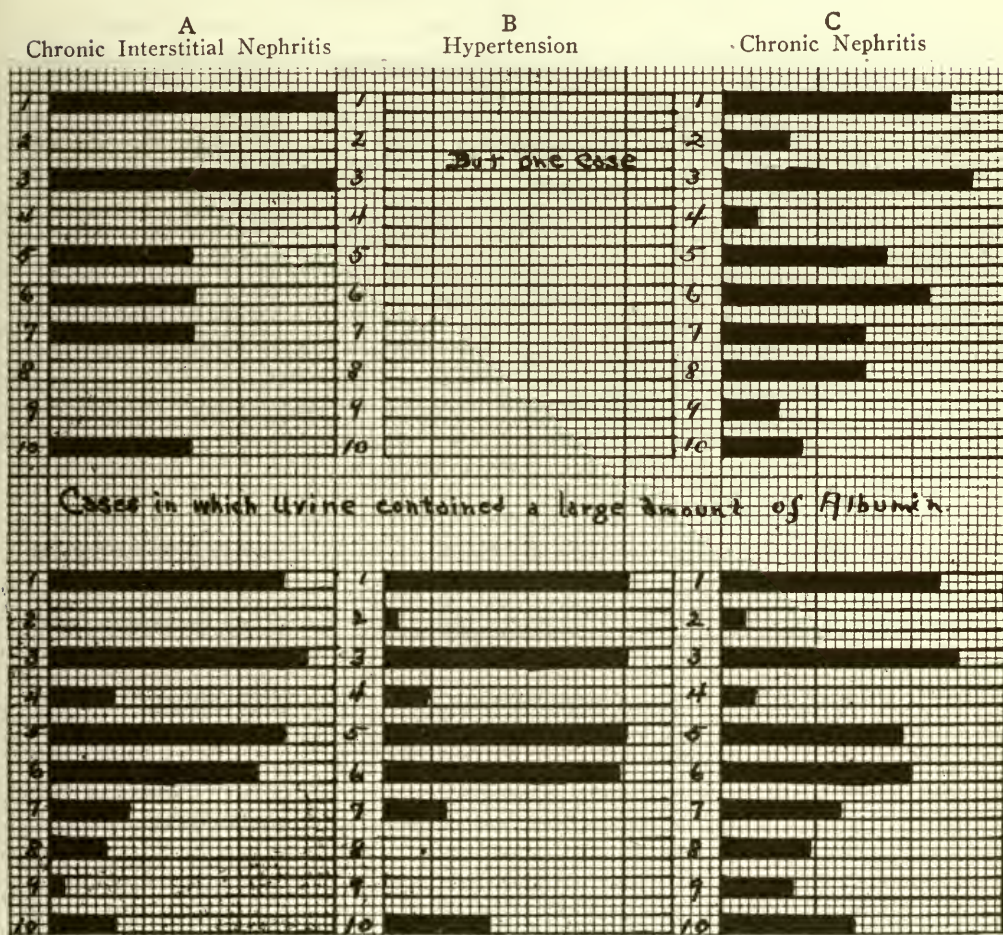


Chart 2. Section 2.—Continued. 1. Edema, congestion or slight swelling of the disc. 2. Swelling of disc 1 D. or more. 3. Arterial changes or tortuosities. 4. Silver wire and corkscrew arteries. 5. Arteriovenous compression. 6. Changes or tortuosities in veins. 7. Hemorrhages. 8. Exudates and other retinal changes. 9. Radiating macular changes. 10. Other macular changes.

amount of albumin was small, but there were but two cases of interstitial nephritis which showed more than a trace of albumin. The other features, Nos. 3, 4, 5, and 6, do not appear to be very much influenced by the amount of albumin in the urine. In interstitial nephritis, their relation to the angiosclerotic process rather than to the amount of albumin found is well shown by a comparison with the graphs showing high blood pressure.

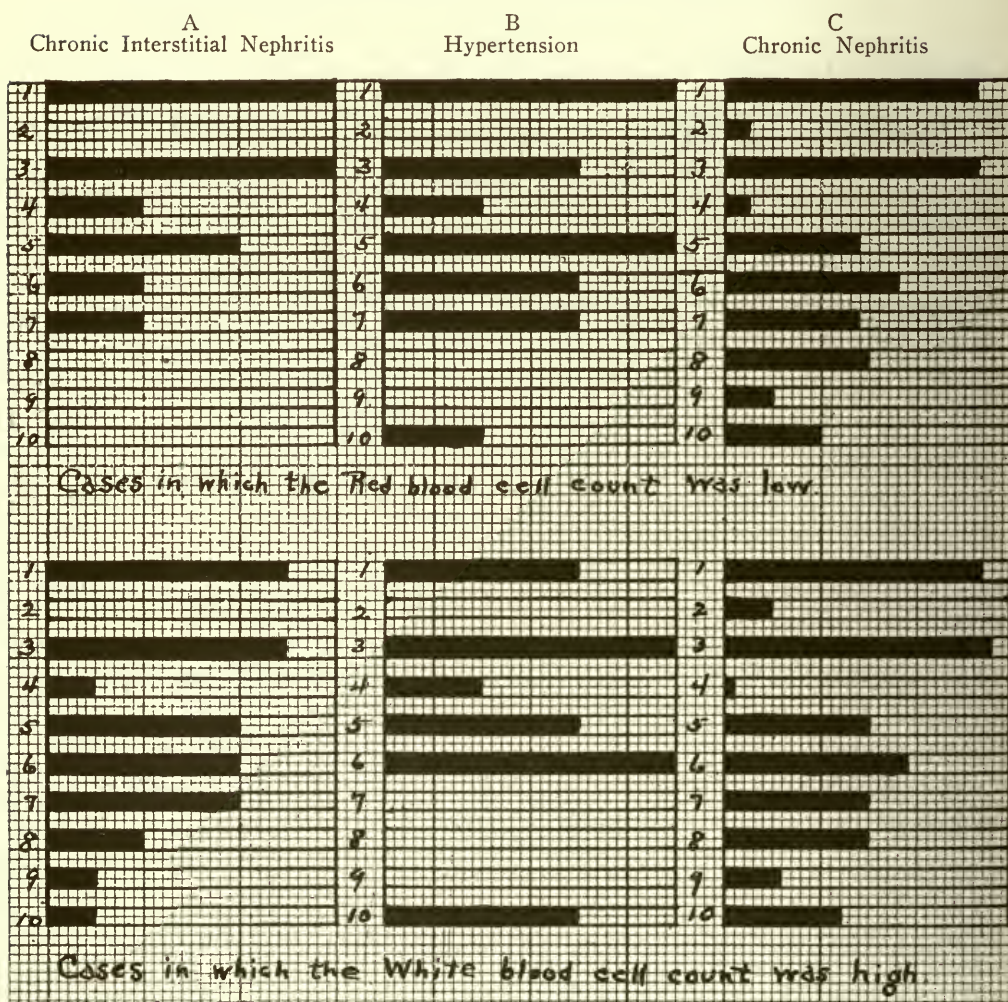
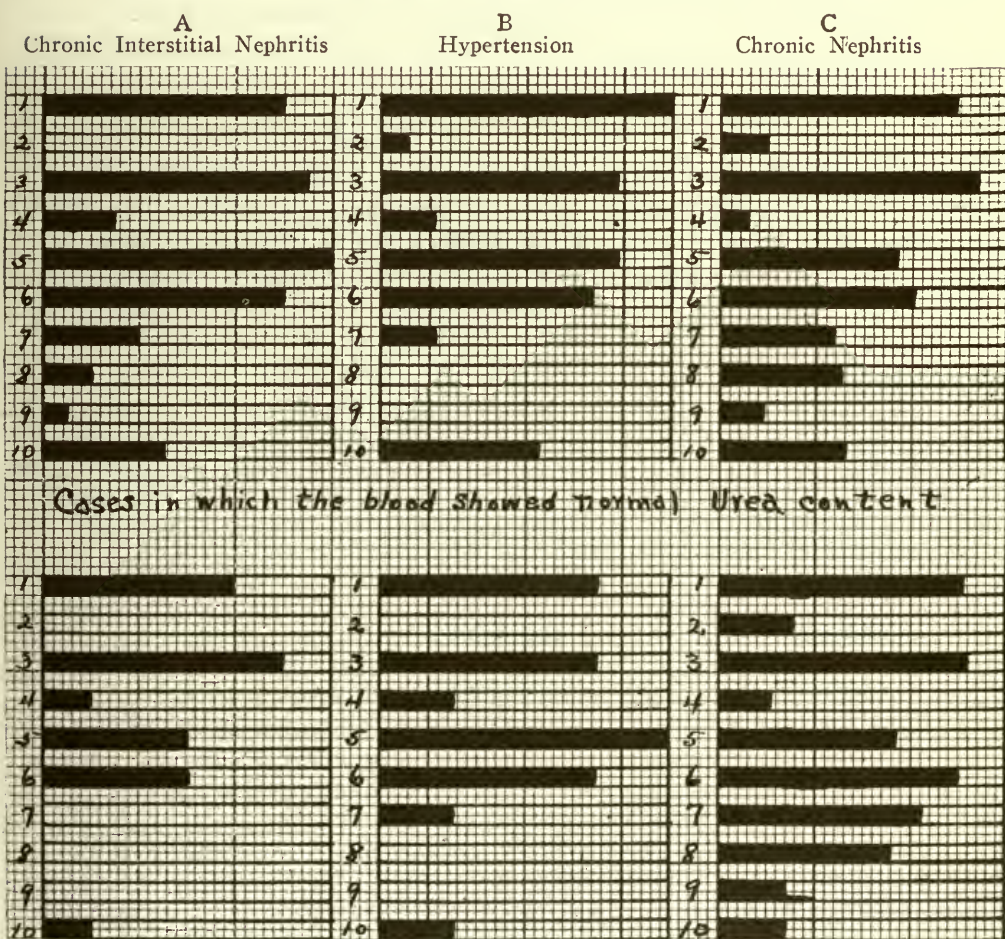


Chart 2. Section 3. 1. Edema, congestion or slight swelling of the disc. 2. Swelling of disc 1 D. or more. 3. Arterial changes or tortuosities. 4. Silver wire and corkscrew arteries. 5. Arteriovenous compression. 6. Changes or tortuosities in veins. 7. Hemorrhages. 8. Exudates and other retinal changes. 9. Radiating macular changes. 10. Other macular changes.

When the blood showed a low red cell count, exudates (No. 8) and radiating macular changes (No. 9) are evidently associated, and to a less degree, swelling of the disc in the parenchymatous group. While vascular features are slightly more prominent in hypertension, they do not seem to be greatly influenced in relative anemia. On the other hand, anemia appears to be a very large factor in the production of the hemorrhages (No. 7), seen in hypertension in this series, and in the interstitial nephritis group in the former series.



Cases in which the blood showed increased urea content.

Chart 2. Section 3.—Continued. 1. Edema, congestion or slight swelling of the disc. 2. Swelling of disc 1 D. or more. 3. Arterial changes or tortuosities. 4. Silver wire and corkscrew arteries. 5. Arteriovenous compression. 6. Changes or tortuosities in veins. 7. Hemorrhages. 8. Exudates and other retinal changes. 9. Radiating macular changes. 10. Other macular changes.

When the white cell count was high, hemorrhages (No. 7) and toxic features, exudates (No. 8) and radiating macular changes (No. 9), were found in both forms of nephritis, hemorrhages being relatively more prominent in the interstitial group and exudates in the parenchymatous group. Marked swelling of the disc was found only in the parenchymatous group of 30 cases with high white cell count. As there were 24 cases in this group with low red cell count, the greater influence of the increase in white cell count over a relative anemia is significant of an inflammatory element in its production, and the same observation applies though in a somewhat less degree, in radiating macular changes. Other macular changes (No. 10) appear to be greatly influenced by a high white cell count; however, too much stress can not be laid on a feature of which there were but two cases.

Increased blood urea was present in about 30% of all cases examined, and in nearly the same percentage in each group. Its influence on the features which appear to be of more or less toxic origin was well shown in the parenchymatous group, in which swelling of the disc, changes in the veins, hemorrhages, exudates, and radiating macular changes are more frequent. In the interstitial group, however, the cases showing no increase of blood urea presented more of these features than when the urea was increased, which perhaps suggests that toxic elements which cause these features are not necessarily dependent upon, although they are often associated with, urea retention. The distinctly vascular features do not appear to vary greatly whether urea is retained in the blood or normally eliminated.

While a considerable number of cases showed cardiac hypertrophy, the only toxic features which appear to be influenced by it were swelling of the disc 1 D. or more, and radiating macular changes in the parenchymatous group, but vascular changes were, by contrast, less prominent. In the interstitial and hypertension groups, the vascular changes for the most part come out quite prominently, especially when compared with the graphs of these two groups for high and moderate blood pressure.

Although a Wassermann test was made in the majority of the cases, only 5 showed a positive reaction. Of these, 3 were interstitial, 12% of the total number, while only 2, or a little more than 2%, gave this reaction in the parenchymatous cases. That it has an influence on vascular changes, exudates and other retinal changes, and on atypical macular changes

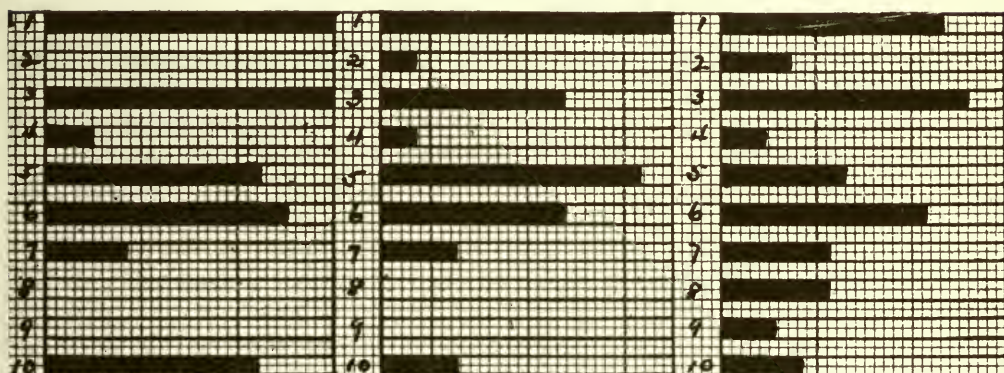
is certainly suggested. However, no definite conclusions can be drawn from so small a number of cases.

As the analysis of the more than 200 cases comprised in the two series has proceeded, the impression has steadily gained weight that neither increased blood pressure, nor the toxic factors which produce nephritis, nor, indeed, any combination of these can adequately explain the peculiar typical fundus features of a late stage of kidney disease. Before offering any hypothesis as a possible explanation of the still

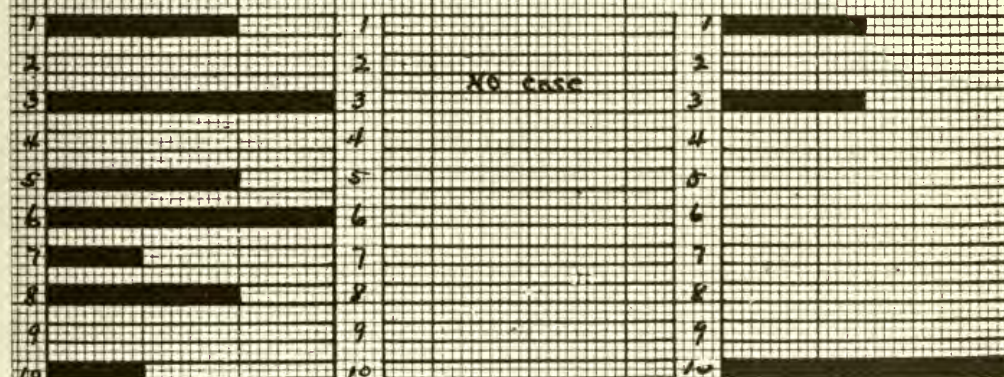
A
Chronic Interstitial Nephritis

B
Hypertension

C
Chronic Nephritis



Cases in which there was present an enlargement of the heart



Cases in which the Blood Wassermann was \pm or +++++

Chart 2. Section 4. 1. Edema, congestion or slight swelling of the disc. 2. Swelling of disc 1 D. or more. 3. Arterial changes or tortuosities. 4. Silver wire and corkscrew arteries. 5. Arteriovenous compression. 6. Changes or tortuosities in veins. 7. Hemorrhages. 8. Exudates and other retinal changes. 9. Radiating macular changes. 10. Other macular changes.

unsolved problem of the etiology of these lesions, may we not turn to the peculiar effects of the internal secretions upon specific physiologic processes and metabolism? That certain ductless organs, for example, the suprarenals, the thyroid, and the hypophysis, exert a profound influence upon these processes, is well established. The effect of ovulation upon the uterus and mammary glands, the effect of lactation upon the uterus and ovaries, the effect of extirpation upon the development of sexual characteristics, the action of the pancreas upon both digestion and metabolism, are examples of a secondary endocrine action in addition to the primary function of these organs. The kidney is an organ having an enormous blood supply. Perhaps no organ of the body is more favorably endowed with the possibility of a secondary endocrine function than the kidney, a theory which has already been advanced by Brown-Séquard. Any material interference with the production of the internal secretion exerts a profound disturbance of physiologic processes. If we cannot satisfactorily explain uremia, or the late fundus changes in nephritis by high blood pressure, albuminuria, increased blood urea, retarded elimination, infection or the products of infection, must we not perforce by analogy suspect that the peculiar later manifestations of the disease, including optic neuritis and the radiating macular changes, are a result of interference with an endocrine function of the kidney, and that such lesions may be correctly described as being nephritic toxic in character?

SUMMARY

In summarizing tables 6 and 7, the following observations may be worthy of note:

First, the average age of both the hypertension and interstitial cases was considerably greater than that of the parenchymatous group, which tends to indicate that interstitial nephritis is not the sequence of parenchymatous nephritis.

Second, high blood pressure is accompanied or followed by vascular changes, which may cause edema and favor hemorrhages and exudates and, perhaps in less degree, certain other features.

Third, while parenchymatous nephritis shows these features also, and several of them more prominently, there appear to be toxic elements which influence the production of optic neuritis, hemorrhages, exudates, retinal changes, and

radiating macular changes, as is prominently brought out by the graphs showing retarded elimination, large amounts of albumin, increased blood urea, and leucocytosis.

Fourth, either the toxic element which accompany the changes enumerated in observation three increases in potency or virulence as the disease becomes more advanced, or some new and more destructive factor becomes active as the function of the kidney becomes more and more impaired.

Fifth, the hope that some distinctive features characteristic of nephritis in its early stages might be found has been disappointed. Nevertheless, it appears to be evident that there are two more or less distinct types of fundus changes, in one of which the features are distinctly vascular in character, and may or may not become prominent early in the disease. The other type, almost invariably late in appearance, is quite as distinctly toxic in origin, and it is because of their intimate association in the late stages of nephritis, when the more typical lesions appear, that the existing confusion concerning the character and significance of these changes has arisen.

The preceding report was made possible by the kind cooperation of Professor L. H. Newburgh and his staff in the Clinic of Internal Medicine, in permitting the use of the cases and in giving access to their records for the reports on the medical findings. Professor Walter R. Parker kindly gave his permission to make the ophthalmoscopic studies, and all the members of his staff have cooperated in making the findings. To Dr. Russell Finch must be given a large portion of the credit for the collection of the notes and the preparation of the tables. For these favors no words of thanks can sufficiently express the appreciation of the author.

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DISCUSSION.

DR. WILLIAM L. BENEDICT, Rochester, Minnesota: The study of retinal changes occurring in the course of nephritis is so complicated, and there are so many factors to be considered, that it is difficult to locate the causative factor and trace it from cause to effect. Furthermore, our knowledge of the subject is undergoing great change, and our literature is no longer up-to-date. For the past fifteen or twenty years, most internists have used the Senator classification of nephritides. For the last four years, and becoming more and more common, internists are using the classification of Vollhard and Fahr. So when we speak

of interstitial nephritis, parenchymatous nephritis, or acute nephritides due to metastatic infection, one should know which classification is used.

As you know, the complications that occur in the retina have been ascribed to two causes. The French speak of azotemia, or the retention of nitrogenous products in the blood, as producing retinitis. Vollhard and Scheik deny this, and claim that the retention of nitrogenous products in the blood has nothing to do with changes occurring in the retina, and that, furthermore, all these changes are due entirely to one single factor, that is, the increase of blood pressure.

I believe, in the later stages of arterial disease, or in older persons in the latter stage of nephritic disease, we have complications that are so interlocked, that it is impossible to separate them as etiologic factors in the production of retinitis.

It is impossible to carry on experimental studies satisfactorily. While we are able to produce a type of nephritis in dogs by the use of mercury, we are never able to reproduce the edema. But edema is common in man, particularly in nephritis which is associated with cardiovascular disease. We know of individuals who have large amounts of nitrogenous products in the blood from obstruction of the small intestines, where the nitrogenous products in the blood will reach as high figures as we have in nephritis. On the other hand, we have individuals with so-called interstitial nephritis, where one kidney is entirely destroyed and the function of the other kidney is carried on with the very small amount of functioning tissue left, and they may go on to complete destruction of the kidney followed by death, without having any marked increase in the nitrogenous products in the blood. So it seems to me that the French school is wrong—that we cannot say that azotemia is the sole cause of retinal changes. We know the German school is wrong—that hypertension and vascular changes are not alone responsible. In Doctor Slocum's series, only a little over 50 percent had high blood pressure. There is no particular relationship between radial arterial pressure and the appearance of the vessels. The amount of kidney destruction is not solely responsible for the changes we find either in the blood or the retina. We can have temporary block of the kidney, which will be accompanied by increase in nitrogenous products in the blood.

The analysis of these cases requires three things: First, a definite conception of what we mean by retinal changes; second, what we interpret as arterial changes, and third, a histologic study of the arteriosclerosis.

DR. RUSSELL L. FINCH, Ann Arbor, Michigan: I was very much interested in Doctor Benedict's discussion, especially with reference to the position of the internist in regard to hyperpiesis and nephritis. In many cases we have a so-called essential hypertension, where no evidence of nephritis is found. We have to come back to the idea of what causes normal blood pressure. Of course we have two factors: First, the force or beat of the heart, and second, the peripheral resistance.

In regard to hyperpiesis in cases with diseased kidneys, there are two main theories, one being the mechanical theory of Cohnheim, which states that the activity of the circulation through the kidney at any moment does not depend upon the need of the kidney for more blood, but rather on the fact that the blood contains nitrogenous material which

must be eliminated, and when atrophy of the kidney is present, the same amount of blood as would pass through the whole normal organ is passing through a smaller area of kidney tissue—thus causing increased pressure. The other theory is the chemical theory, and I think needs no discussion, except to say that there are supposed to be pressor substances secreted by the suprarenals and the kidneys which cause hypertension.

I think it is important, that in many of these cases of hypertension we do not find evidences of nephritis; in many we do not find renal lesion. Even with a blood pressure of 230, or higher in some cases, we have no renal lesion or evidence of nephritis. We have hypertension, but no albumin, normal specific gravity, and in many cases no cardiac complications.

The old classification of nephritis into chronic interstitial and chronic parenchymatous is, I think, still true from a clinical standpoint. We can find certain cases that fall into the class of chronic interstitial nephritis. In such a case, the patient is usually beyond middle age; they nearly always have heart complications, with probably some evidence of cardiac failure; they have well marked arteriosclerosis; very low specific gravity of the urine; little or no albumin; and the prognosis we know is good probably for years. They may carry on a fairly normal existence without handicap. They may or may not have hypertension. But we have also the other cases of chronic parenchymatous nephritis, usually in younger people, which often comes on insidiously. They usually have edema; they may or may not have hypertension; usually large amounts of albumin are present in the urine, and there may be present well marked retinal changes outside of the sclerosis. Many people say you cannot differentiate chronic nephritis into the interstitial and parenchymatous types, but that discussion probably will go on for some time. But in the cases we have seen, we can definitely classify them into chronic interstitial and chronic parenchymatous nephritis.

DR. GEORGE SLOCUM, Ann Arbor, Mich., (closing): I would like to have you follow these charts through with me, noting the marked difference in the length of the different graphs in chronic parenchymatous and chronic interstitial nephritis. Again, note that the average age of the cases of chronic interstitial nephritis and hypertension is something like ten years older than the average of chronic parenchymatous nephritis in our series. Does not this show that chronic parenchymatous nephritis is not a late stage of chronic interstitial, nor primarily a result of vascular change? Many of you are familiar with the difference in the clinical appearance of the two types of disease, and have been struck by the outstanding difference between cases that die in middle life from chronic parenchymatous nephritis, and those who have chronic interstitial nephritis and live to be old and die of an intercurrent disease. Does not this likewise show that chronic interstitial is not a later stage of chronic parenchymatous nephritis? While histopathologic sections often assist in the classification of kidney disease, they frequently fail to improve on the clinical classification, or to explain satisfactorily the clinical manifestations of the disease.

SMALL-POX KERATITIS.

JAS. M. PATTON, M.D., F.A.C.S.

OMAHA, NEBRASKA.

Small-pox, as recorded in the early history of central and southern Asia, gradually spread until it became an almost universal scourge. At times, it even surpassed bubonic plague as an enemy of mankind, for it not only killed countless thousands, but those who recovered were often horribly disfigured, and many were blinded for life.

Against this enemy of the race, the medical profession stood helpless. Human inoculation had been practiced by early Asiatics and was introduced with the disease into Europe. Later "small-pox purchase" as it was called, or the exposure of a healthy individual to a mild case of small-pox in the hope that a light infection might produce an immunity was tried, but both methods were uncertain, and too often, instead of producing immunity, resulted in the death of the patient. Not until the introduction of vaccination in 1798 by Jenner, was there any real promise of relief from this world wide scourge. If the enemies of vaccination would take the trouble to compare the post- and prevaccination history of small-pox, they would at least have to admit the absurdity of their contention.

With the decrease in the virulence and extent of small-pox, the accompanying involvement of the cornea has promptly diminished, but it is our impression that this complication occurs more frequently than is generally supposed, and that the oculist may be of service in calling this to the attention of members of the medical profession.

The literature on this subject is not as full as might be expected. Scarpa in his monograph on Diseases of the Eye, published in 1801, reports the case of a girl of 1½ years, who after a severe case of small-pox had a small grayish ulcer, the size of a millet seed, on the nasal part of her cornea, and a small beginning abscess near the upper limbus. The ulcer healed after touching it with copper sulphat, the mother washing the eye with milk. The abscess remained stationary for a time, but later the surface broke down forming an ulcer similar to the first. It was treated twice with copper sulphat

crystal and also with a copper sulphat collyrium. The eye was healed after about six weeks, with a dense corneal scar.

Jonathen Hutchinson writing on "miscellaneous cases" in Moorefields' Ophthalmic Hospital Reports, 1856, page 333, states that "inflammations of the eye not infrequently occur in patients who have suffered from small-pox, but who are quite convalescent before the eyes are attacked." He describes "a central, rather superficial ulcer which may spread," and calls attention to the inexcusableness of confusing it with "scrofulous ophthalmia." There were no evident age limitations in his cases. The condition was always unilateral and treatment was unsatisfactory. On page 369 of the same report, he reviews an article by Mr. Marson, "our chief English authority on all which relates to small-pox," who stated that, in 1838, out of fifteen hundred cases treated, he had not seen the formation of a pustule on the eye, and in the observation of fifteen thousand cases treated at the small-pox hospital, he had never seen an eye injured as the result of the disease. Hutchinson explains this very favorable report on the ground that ocular symptoms usually come on after convalescence, or after the patient had in all probability passed out of Mr. Marson's observation.

He further observes that corneal involvement is more likely to occur in the presence of a high secondary fever with unusual dryness of the skin, and in virulent cases, when the destruction of the cornea may be very rapid.

Nettleship in Curator's report, Moorefields' Ophthalmic Hospital Report, 1873, page 208, 585 and 586, gives the pathologic findings in four eyes removed as the result of small-pox. In the first there was marked staphyloma with thickening of the central cornea. No clinical history given. The second was a child of four years, where the eye became sore immediately after the eruption of the small-pox. There may have been a secondary injury. The lower half of the cornea was staphyломatous and much thickened, with adhesions. In the third case, a boy of 7, the corneal infiltration came on during the eruptive stage. At the time of the operation, ten months later, the cornea was uniformly opaque and staphyломatous. The keratitis did not come on in the fourth case, a boy of 12, until after he was up and about. On admission to the hospital, the cornea was irregular, thin and bulging.

These evidences of destruction are more severe than we have seen in our practice. On the other hand, it would only

be the extreme cases that would likely apply for hospital treatment sixty years ago.

Statistics as to the results of this condition in the United States, though not conclusive, are at least of passing interest. Best, in his excellent work "The Blind," page 103, under the head of General Diseases causing Blindness, gives small-pox 0.8%, which considering the preventableness of this disease, seems to us to be rather high. The Census Bureau reporting on the blind of the United States up to 1910, gives the total number of individuals blind from small-pox as 227, being the 0.8% of the entirely blind population of the United States as reported by Best; this only includes those whose vision is so far diminished that they can not read with or without glasses, and does not take into consideration the large number who have lost one eye, or who have one or both eyes partially crippled from keratitis of this origin.

Comparison with European statistics is unsatisfactory. A table in the above mentioned report gives Austria in 1906, 841 cases with a percentage of 5.2; Bavaria in 1903 had 24 cases with a percentage of 0.7; Ireland in 1911 had 71 cases with a percentage of 1.6; Switzerland in 1896 had 43 cases with a percentage of 1.2. However, Professor Ernst Fuchs, during his recent visit to America, made the statement that owing to the extreme enforcement of vaccination in Vienna, small-pox and its complications including those of the cornea are practically unknown. This in spite of very unsettled sociologic conditions. Compare this with Mexico, where vaccination is not compulsory, and where according to Ruiz, (*Zentralblatt für die gesamte Ophthalmologie*, February 14, 1922, page 24) 20% of blindness is due to small-pox.

Of Americans reporting recently, Decker, *American Journal of Ophthalmology*, volume 4, page 854, presents two cases. In one, a man of 24 years, the ocular irritation and secondary small, superficial nonstaining spots of corneal infiltration appeared three or four days before the skin eruption. The second, a woman of 52, showed iris irritation and corneal infiltration during the eruptive stage, but the cornea did not break down until two months later. This continued to advance until the corneal defect was thoroughly curetted. Smith, *American Journal of Ophthalmology*, volume 5, page 32, reports three cases of keratitis, and Burnam in the same volume, page 123, one case. Smith's cases were young people, and in each the corneal lesion came on one or two months

after the small-pox had subsided. Two of these had been previously diagnosed as interstitial keratitis. In spite of the most approved and energetic treatment, two of the eyes were lost, and the third recovered with reduction in vision, after drainage of the anterior chamber by a Saemisch incision. Burnam's case was a young man of 33, who had had small-pox two months before consulting him. There was a large dense infiltration of the outer half of the cornea, with haziness of most of the inner half. He reported a final vision of 6/9 on the combined treatment of Lewis, which he considers of very great value in corneal infections.

Dr. W. G. Gillett, Wichita, Kansas, in association with Dr. J. G. Dorsey, has kindly sent me the notes of ten cases which they have seen in their practice in the past few years. The ages of their cases were from six weeks to 43 years, three females and seven males. In all, the corneal complications developed in from two to eight weeks after the general disease had subsided, the average being about six weeks. In eight of these cases, a sharply outlined, disc shaped, cloudy opacity was present in the deep interstitial tissues, the process very closely resembling disciform keratitis. There was marked congestion of the ocular conjunctiva. Focalized light showed no superficial ulcerations, nor was there any evidence by staining with fluorescein. The treatment included the use of atropin, dionin and heat, and all were very slow to clear up, the convalescence lasting anywhere from two months to two years.

Going back over a period of something less than five years, I find that we have records of 36 cases treated for keratitis associated with, and undoubtedly the result of small-pox. Of these, 30 cases were males and 6 females, the oldest past 60, and the youngest covered with small-pox vesicles at birth. The largest number of cases seen in one year was ten in 1921, the smallest, three in 1919. In one case, the keratitis appeared before the general eruptive manifestations, one had small-pox at birth with probable corneal involvement, sixteen during the eruption, and the remaining eighteen after the general symptoms had partly or entirely subsided.

The lesions were generally central in 14 cases. Usually circular, rather deeply infiltrated, more gray at the center, and staining, if at all, over only a portion of the area. In twelve cases, they were peripheral, located within 2 to 3 mm., of the limbus, and were apparently not so deep as the central type.

In three cases, the infiltrates were definitely crescentic in shape; six were diffuse and hazy in appearance, without much difference in the density of the infiltration. There was considerable loss of corneal tissue in five cases, one of these requiring evisceration some six months later on account of the persistent irritation and discomfort.

The congenital case was of rather unusual interest, as both parents were recovering from small-pox, the mother still having a few unhealed vesicles when the child was born. There were numerous vesicles on the baby's face, scalp and limbs, and it carried a temperature of 102° Fahrenheit for two or three days after birth. When seen by us six weeks later, the left palpebral fissure was smaller than the right, and the left cornea was relatively smaller and somewhat hazy. At the age of six weeks, the child weighed only 5½ pounds. To rule out the possibility of lues, a Wassermann of the mother's blood was taken and reported negative. Another case, seen at the age of three months, had small-pox when nine days old. There was a rather thin opacity slightly overlapping the center of the right cornea, but the eye was free from irritation, with the promise of gradual improvement. A third infantile case seen by us, at the age of six months, had small-pox about a week after birth. The right eye was sore for several months, but was practically free from inflammation for a month before our examination. We found a rather dense central opacity of the right cornea, reaching to within 3/32nds of an inch of the limbus in all meridians.

I mention these infantile cases, as it is so easy for the attending physician to overlook a corneal involvement in the very young, especially if the lids and face are much involved.

Our case that went to evisceration, when first seen showed a diffuse nonstaining infiltration of the entire right cornea, the keratitis appearing during the eruptive stage. Improvement was slow, but the eye became quiet and remained so for six months. Then the patient returned with a densely vascularized corneal scar, more or less discomfort, and a tendency for the ocular inflammation to flare up on slight provocation. About one-third of the cornea with a piece of iris adherent was excised at the operation by my colleague, Dr. Sanford R. Gifford, who examined it microscopically and reports as follows: "The specimen comprises about one-third of the cornea with a piece of iris adherent to it. The epithelium is very rough and thrown into numerous small folds,

much thinner than normal in some places and much thicker in others. Bowman's membrane is absent in many places. A very active cellular infiltration is seen throughout all layers of the cornea. Numerous spaces in the parenchyma are full of large and small mononuclear and epithelioid cells. New vessels are seen through the cornea and surrounded by mononuclear infiltrations. Many of the cells in the infiltration are

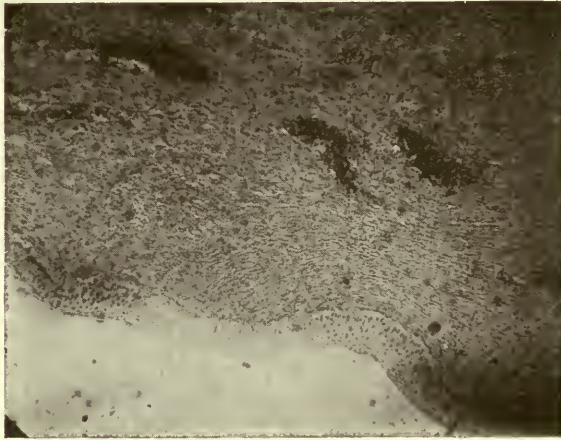


Fig. 1. Low power, showing irregular epithelium and areas of round cell infiltration.

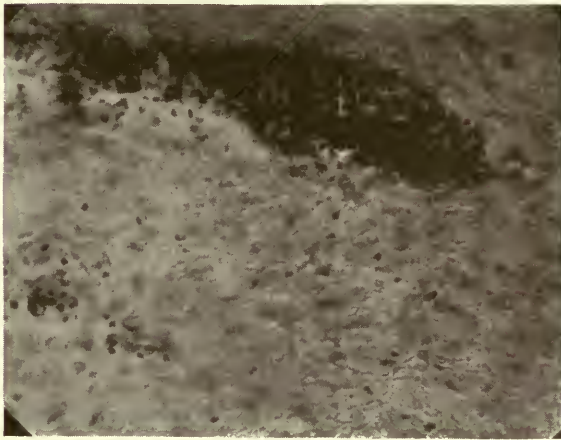


Fig. 2. High power, showing irregular areas of round cell infiltration.

Microphotographs of the cornea and iris in a case of small-pox.

true eosinophiles. The infiltration in the portion of the iris present is similar to that in the cornea. This specimen is of special interest, as it shows how even after six months, in an apparently healed cornea, active inflammation is still present in spite of almost continuous treatment." The general resistance of this patient was not good and may have accounted in part for the unfavorable outcome. (Figs. 1, 2, 3, 4.)

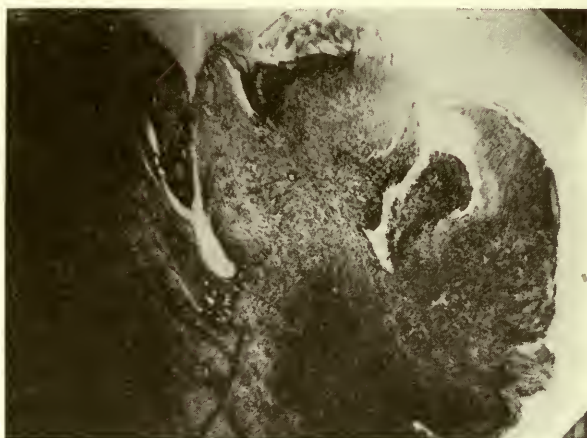


Fig. 3. Low power. Dense infiltration at point of fusion between cornea and iris.

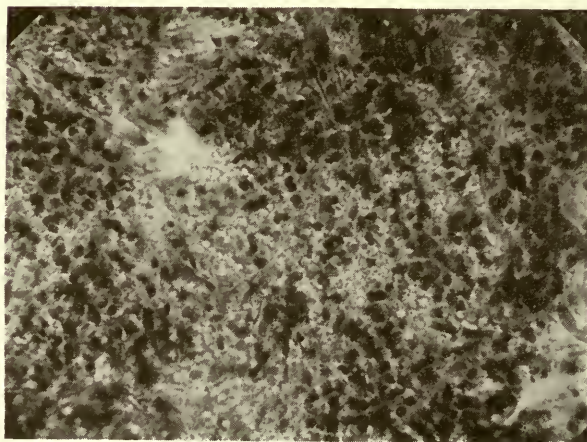


Fig. 4. High power. Dense infiltration at point of fusion between cornea and iris.

Microphotographs of the cornea and iris in a case of small-pox.

B. W., age 47, was a little out of the ordinary, in that his infiltration appeared about a week before the active eruption. The eye had been feeling irritable for about a week before this infiltration appeared, and as he had been exposed to small-pox, he was vaccinated at about this time. There was a satisfactory vaccination vesicle, but this did not prevent the appearance of small-pox about ten days later. The cornea showed a small infiltrate near the upper margin, with a hazy area extending down below the center. He was unable to stay for treatment at his first visit, but we wrote directions to his family physician, who after ten days reported no progress, so he was returned. On his second visit to the office, the eye was still deeply congested, with very slight hypopyon. The actual cautery was used, followed by daily applications of iodine. He improved very promptly, and a letter from his family physician, three weeks later, reported complete convalescence, but with no mention as to the amount of his vision.

The following, our only bilateral case, shows the importance of a guarded prognosis. The patient, a man of about 55, had small-pox a month before coming to Omaha. His eyes began to be affected about two weeks previously. Both eyes showed a moderate deep congestion, with a subepithelial ring of infiltration about $3/16$ ths inch across. The ring of infiltration was about $1/16$ th inch wide and surrounded a central area of clear cornea. The infiltrate showed very slight roughness on the surface, but did not stain under fluorescein. Vision about 20/50 each eye with correction. In spite of hot applications, a collyrium and atropin, the clear portion of the center within the ring gradually became opaque, and the surface broke down over an area about $1/16$ th inch in diameter. This took several weeks to heal, and the patient left for home with his sight very much worse than when he came. Approximate vision not more than 15/200. The final outcome is not known.

Dr. Sanford Gifford examined a few of these cases with the slip lamp and corneal microscope. It was seen that the denser parts of the opacity extended rather deeply into the cornea. At the advanced portions, however, delicate feathery lines could be seen in the more superficial layers, just under the epithelium, so that apparently progress was being made in this layer first.

Improvement in these cases is usually discouragingly slow. Some remain stationary until the deep grayish spot of infiltration is burned out with the electric cautery. The application of heat with Shahan's thermophore at about 140° F., seems to help in some instances. Chemical cauterizations, for example trichloroacetic acid, nitric acid or iodine, is indicated in the open cases. Drainage of the anterior chamber by a delimitating keratotomy in the progressive cases, or a Saemisch incision in the indolent ones, is of especial value. If the corneal incisions close, as they frequently do, they should be reopened at least twice a day to get the best results. If there is marked congestion with iris involvement, the use of atropin and salicylat is indicated, with repeated applications of moist heat. Intramuscular injections of boiled milk relieves the accompanying pain in most instances, and may be of value in hastening recovery. At best, these cases are slow to respond to treatment. This should be carefully explained to the patient at the start. When the condition of the cornea will warrant it, we have the patient return at once to his family physician, whom we supply with careful directions as to treatment and a request for a report as to progress. If there is any doubt as to this, the patient is asked to return for re-examination. A guarded prognosis as to recovery of vision is advisable, though as a rule the eye becomes quiet after some weeks and remains so. Physicians treating active small-pox should be on the outlook for the beginning of ocular symptoms. Treatment should be instituted as early as possible, and even when the process is extensive, vigorous therapeutic measures may save clear cornea and useful vision to the patient.

DISCUSSION.

DR. NELSON M. BLACK, Milwaukee: As Doctor Patton has mentioned, before the advent of vaccination, blindness was frequently a sequela of variola. Parsons states that blindness occurred in from 1.3 to 2.5 percent of all cases, and that of the diseases of the eye caused by smallpox, 38.78 percent are affections of the cornea. They never occur before the tenth day, generally from the twelfth to the fourteenth day, and the corneal involvements are small, circumscribed infiltrations, interstitial keratitis and hypopyon ulcer.

It has generally been accepted that the keratitis is not due to pustules, and that the severity of the corneal complication is not always proportionate to that of the general disease. Self infection from vaccination is rare, but does occasionally occur, one eye only being affected. The cornea is very rarely affected primarily, although two or three undoubted

cases have been reported. The keratitis, if any, is an extension from the lids or conjunctivae.

"Since the *ulcus serpens* in variola develops such a length of time after the stage of eruption, it obviously cannot be regarded as a small-pox pustule that has been localized upon the cornea. Such pustules do occur on the eye, but only in the conjunctiva. It is true they may then, if they lie near the margin of the cornea, give rise to a suppurative infiltration of the adjoining portions of the latter, but not to the typical picture of the centrally situated *ulcus serpens*. When the latter develops in the stage of desiccation of variola, it must be attributed, like a traumatic *ulcus serpens*, to an infection of the cornea from without. There is no lack of opportunity for such infection to take place, since the free border of the lids is a favorite seat for variolous pustules, which thus can come into direct contact with the cornea."

Smallpox keratitis, then, in the absence of a lesion of the lids or conjunctiva, must be of endogenous origin, either from bacteria brought directly to the cornea or, by toxins carried in blood or lymph streams. The fact that the corneal involvement does not make its appearance until the stage of desiccation, or even until patients are up and about, would indicate that the cases of primary corneal involvement were endogenous infections.

The length of time which lapses in the course of the disease before the keratitis manifests itself may be accounted for by Leber's biologic theory, i. e., in the normal cornea there is only a small number of antibodies, but during the active stages of inflammation of this nonvascular structure, conditions are brought about which encourage the admission of antibodies into the corneal tissues. Fuchs, by inoculating the cornea of animals with various pyogenic organisms, has explained, as a result of these experiments, how infectious suppurative keratitis is produced. Following the inoculation, the cocci multiply in numbers, and the cornea, for a certain distance surrounding the point of inoculation, perishes as a result of the powerful toxic substances excreted by the germs. Leber believes that substances produced by the bacteria exert an irritant action upon cell protoplasm when slightly concentrated, but when possessed of strong toxic properties they paralyze the cells. The result of the former is to attract the cells toward the area of inflammation, a phenomenon known as a positive chemotaxis, which may be explained by the action of the aggressins [bodies supposed to inhibit the paralyzing properties of aggressinogens on the leucocytes], while the effect of the latter is to paralyze the cells and render them unable to reach the inflammatory focus, a condition known as negative chemotaxis, for which the aggressinogens are probably responsible. Clinical examples of negative chemotaxis are occasionally seen in the early stages of corneal infections, in which there is a zone of clear cornea interposed between the infected area and the ring of infiltration.

Councilman has shown, that fifteen minutes after central infection of the cornea with staphylococci, granular leucocytes are found in the conjunctiva; these are also the first to appear in the cornea, and form the majority of the wandering cells. In eighteen to twenty-four hours, isolated nongranular leucocytes are found in the peripheral part, and in four days, lymphocytes. The latter occur only in the lymph channels

at the periphery, and probably come from lymphatic glands and not from the blood. After five days, plasma cells appear in the outer third of the cornea. Thus considerable time can be accounted for before indication of active inflammation in the cornea would be noticed.

The long duration of smallpox keratitis and the slow recovery is undoubtedly largely due to the nonvascularity of the cornea. The first evidence of repair is seen in the development of new vessels from the part of the limbus lying nearest to the focus, or, when this is central, from the whole periphery. Their chief function is that of supplying the necessary material for making good the loss of substance. They lie in the most superficial layers of the cornea, between the epithelium and Bowman's membrane; but in severe or prolonged inflammations, they are also formed beneath Bowman's membrane. Other signs of regeneration are seen in the mitotic division of the corneal corpuscles, from which the scar tissue is formed, though the plasma cells may take part in the process.

Under treatment, the thought has suggested itself, inasmuch as the progress and healing process are so slow, that in addition to local treatment, some internal medication directed toward the supplying of the "something" lacking to stimulate regeneration of the lost tissue might be effective. Possibly strychnin or some endocrine therapy might be efficacious, as has been used with success in the treatment of low grades of uveitis.

Evidently Doctor Patton has had the opportunity of seeing many more cases of smallpox keratitis during the last five years than many of us in special work.

DR. J. G. DORSEY, Wichita, Kansas: There are two things that attract your attention in these injuries. One is that they begin late. In only one instance have we seen a case during the active stage of the fever. In most cases it comes on late. The other point is that they do not clear up quickly. Due to the fact that Doctor Black has mentioned—the lack of ability to clear the cornea—it seems to take a long time for the lymph channels to empty themselves, and I have frequently seen it as much as a year afterwards before active inflammation had entirely subsided.

DR. JAMES M. PATTON, Omaha, Neb., (closing): I particularly wish to emphasize the use of anterior chamber drainage in these cases. In addition to regular routine treatment, anterior chamber drainage seems to afford more relief than any one thing.

Another thing is, that we should impress upon our colleagues in general practice the importance of being on the lookout for this condition in their cases of smallpox. They are very slow and discouraging, but at the same time the earlier the treatment is instituted and the more carefully it is pursued, the better chance these patients have for useful vision.

UNERUPTED AND IMPACTED TEETH CAUSE OF
SERIOUS EYE AND EAR CONDITIONS.
PRELIMINARY REPORT.

J. A. STUCKY, M.D.

LEXINGTON, KENTUCKY.

Notwithstanding dental examination with the X-ray of the teeth is a routine in many of our diagnostic clinics, on account of the fact that the examination goes only as far as the alveolar process and the roots of the teeth, these frequently do not give us the cause of the trouble, if it happens to be, as in the cases to be presented, above the alveolar process. Only three cases out of eight will be cited, because these typically illustrate the conditions found in the others. In all of these cases, all evidence of focal infection was removed. This included blood tests, Wassermann and spinal fluid, gastrointestinal examination, and examination by the internists and neurologists. The cases were referred back to me after careful examination and observation of several months or years.

CASE 1. A woman of twenty years, whose left eye was amblyopic from birth, came suffering with irritative iridocyclitis; this was relieved by the usual treatment of hot applications, rest in bed, purgatives and sedatives. Later, she came with the clinical picture of acute glaucoma of a fulminating type. Tension was markedly increased, cornea hazy, pupil irregularly dilated, anterior chamber shallow. For this an iridectomy was done. This gave immediate but temporary relief. In a few days all of the symptoms of iridocyclitis and glaucoma returned. At this time, the patient begged that her eye be removed. A stereoscopic roentgenogram was made, which revealed an unerupted wisdom tooth on both sides. These were extracted and the patient had immediate relief. It has been more than a year since the operation, and there has been no return of any of the previous symptoms.

CASE 2. A female, aged twenty-eight, of a neurotic temperament, had been constantly under the care of surgeons and physicians for the past seven years. During this time, the appendix and thyroid had been removed. These operations improved her general health, but she continues to have head-

aches with tachycardia and is unable to use her eyes for close work for any length of time. She has been repeatedly refracted by different oculists, with only temporary relief. The refractive error consists of hypermetropic astigmatism. She stayed four months in a sanatorium, during which time she received the pluroendocrine treatment, and this relieved her nervousness and tachycardia, but she still suffers with headaches. This patient had also had two dental examinations and X-rays made, with negative results. Encouraged by the first case, stereoscopic plates were made, which revealed unerupted wisdom teeth on the right, and an impacted wisdom on the left. These were removed, and all of her symptoms were relieved as if by magic.

CASE 3. A woman, thirty-nine years of age, mother of two children, suffered with discharging ear in childhood; her general health has been excellent until four or five years ago. Since then, she has been nervous, irritable, suffering frequent pains deep down in the ear and shooting pains over the temple and back of the ear. Examinations of blood, secretions, gastrointestinal tract gave no evidence of focal infection. The drum membranes were normal except for cicatrix in the left. There was no evidence of inflammation or suppuration. Blood count and blood test normal. Notwithstanding she gave the classical clinical symptoms of chronic mastoid involvement, there was no redness or swelling of the parts—nothing but deep seated, intense, lancinating pains—no pain on deep pressure. An X-ray of the mastoid was negative. Diagnosis was made of neurotic mastoid, and the patient referred back to the internist. After six weeks, the case was again referred to me, and I had a stereoscopic X-ray made, which revealed unerupted lower wisdom teeth; these were extracted and all of this patient's symptoms were immediately relieved.

The nervous manifestation or pain occasioned by impacted wisdom teeth is practically never manifested at the point of impaction, but somewhere else. It is most often found in some other branch of the trigeminal nerves, but it may be shown by reflex pain or disturbance in some other part of the body. This may explain cases of headaches, eye irritation (ophthalmic branch trifacial), pains in lower jaw, (mandibular branch of the trifacial).

Believing that it should not be necessary to have stereoscopic plates made in these cases where we excluded every cause of the clinical symptoms but the teeth, I consulted an expert roentgenologist, and asked him why dental plates made in all of these cases and their interpretation were negative, although the cause of the trouble was just below or above the teeth. He suggested the following method of making the plate in dental examinations:

"With the patient comfortable in the chair, with the grinding surface of the teeth on a plane parallel to the plane of the floor, place the film in a position against the hard palate, in case of upper teeth, with the anterior border of the film at the middle of the first molar. The lower border of the film is even with the grinding surface of the molars, and the film is held in place by the finger of either the patient or operator. The angle formed with the perpendicular plane by the plane of the film, must be bisected with a plane (imaginary), and the X-ray instrument directed perpendicularly upon this imaginary line, in order to take a true picture."

These three cases have been under observation from one to two years after the removal of the cause of the trouble, and there has been no return. In a future report, I will present the other cases referred to.

DISCUSSION.

DR. EDWARD STIEREN, Pittsburgh, Pennsylvania: When one considers the wide distribution of the trifacial, and remembers that it has a threefold function, sensory, motor, and (through its connection with the ciliary ganglion) sympathetic, it is not surprising that these affections due to impacted teeth occur.

I have never seen any case such as Doctor Stucky reports, due to impaction. I have seen a number due to focal infection from teeth. Two impaction cases I now recall—one a girl twelve to fourteen years of age, who had an intractable punctate keratitis. No treatment seemed to benefit her until we had an X-ray picture of the mouth, and found an impacted molar on one side. It was almost miraculous the speed with which the spots cleared up after the molar had been removed.

Recently I had a similar case, a young man of thirty years, who presented almost the same picture of punctate keratitis of the upper layers of the corneal tissue proper. Remembering my experience with the other case, an X-ray was taken of his mouth, and an impacted molar found on the same side. Extraction of this tooth was followed by subsidence of the keratitis.

DR. ARTHUR G. BENNETT, Buffalo New York: I want to record two cases that I have seen in the last three or four years,

The first was the assistant superintendent of the hospital with which I am connected, who had complained of inability to use her eyes for more than a few minutes. Her duties called for the use of her eyes most of the time, and she was greatly handicapped. I refracted her eyes several times, and had her gone over by an ear, nose and throat man, and also by a general practitioner, and their reports were negative. At last it was suggested that she see a dentist. He made an X-ray and found two unerupted wisdom teeth. The removal of these teeth immediately cleared up her vision.

The other case was a nurse in the hospital, a young woman about twenty-one, who came complaining of loss of vision in the lower field. We found perfect hemianopsia of the lower field in right eye, and at that time there was a little papillary edema. The next day the hemianopsia had become three-fourths, until finally the eye was totally blind. A fine internist saw her and went over her carefully, including a Wassermann. Nothing was discovered until he suggested the teeth, and the dentist found two unerupted wisdom teeth, impacted. With their removal, the symptoms subsided and she recovered with fair vision. The optic nerve, due to the intensity of the edema, had evidently been affected, and she has some atrophy. But she has 6/9 vision.

TRAUMATIC FACIAL DIPLEGIA, WITH INVOLVEMENT OF THE SIXTH NERVES, PORTION OF THE LEFT THIRD NERVE, ALSO THE FIFTH AND EIGHTH NERVES, WITH DISLOCATION OF THE ATLAS, ETC.

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Traumatic facial diplegia is apparently a very rare condition. The great war produced but very few cases. A survey of the literature of this subject for the last thirty-five years, made with the help of the librarian of the Office of the Surgeon General of the United States Army, has brought to light the few cases on record, none of which parallel in all respects the description of the case which it is my privilege to present to you to-day, a case which is of interest to the ophthalmologist, oto-laryngologist, neurologist and general surgeon, alike.

Bilateral facial palsy may be due to a variety of other causes. Patrick gathered together forty-eight cases due to multiple neuritis. Ross found five due to diphtheria, and five due to influenza. It may be due to double otitis media and mastoiditis. Throckmorton reported a case due to rheumatism, wherein an interval of a week elapsed before the other side of the face became involved. Sir Charles Bell reported a case due to syphilis. But the cases due to traumatism are indeed very rare.

Ransohoff, in a communication to the 1919 meeting of the American Surgical Society, could find but four other cases in the literature than his own, and appended one with Sach's permission, hitherto unreported. He failed to note the earliest case, reported by our own lamented confrere, Alvin W. Hubbell, of Buffalo. In giving a resumé of the cases reported to date, I quote liberally and literally from the writings of the reporters.

The first case to be reported, as far as we can find, as intimated above, was in 1889 by Hubbell: Henry M., aet. 23, a switchman, while coupling cars, August 21, 1888, had his head caught between the projecting ends of the loaded lumber on two cars, between which he stood at the time. He was picked

up unconscious and hurried to the hospital. Hemorrhage poured profusely from both ears, nose and mouth. He vomited blood frequently. Pulse was 54. Diagnosis was given as fracture of the base of the skull. On the following day he was in a semiconscious state. On recovering consciousness, it was found that the hearing of both sides was somewhat impaired. Complete facial paralysis of both sides of the face was noted. The patient was unable to close the eyelids, or to move the lips. His face was absolutely expressionless. Both right and left abducens were completely paralyzed, resulting in a marked convergent squint of both eyes. The pupil of the right eye was dilated, and its accommodation impaired. The right side of the tongue was dry and rough, with total loss of the sense of taste. Deglutition was difficult. Sensation and motion in other parts of the body were normal. He had intense pain in the forehead. He gradually improved, and his sense of taste returned after a month. The sight and hearing returned to normal. The pupil contracted down. The reporter, on July 2, 1889, stated that the facial paralysis had so far improved, that he could nearly close his eyes, and in laughter could move some of the muscles of the mouth. The left abducens had pretty nearly recovered its function. The right abducens was still paralyzed.

For the following four case reports, I am indebted to Ransohoff, in the article mentioned above.

"CASE 1. (Koslovsky). Private J. J., white, while carrying logs from a boat to a landing, fell into the water, a distance of ten feet, and was struck by a log over the left side of the head. When brought to the hospital, he was bleeding from both ears and nose and was vomiting. Consciousness was retained, but speech was very indistinct. There was a large hematoma on the left side of the scalp. No other external injuries. He had considerable headache. Temperature was normal, likewise the pulse. The pupils refused to respond normally to light and shade, but later became normal in the right eye. The left pupil was dilated and did not respond to light. There was constant and marked diplopia. Hearing was much lessened on the right side, and entirely lost on the left side. The tuning forks could not be heard on this side. There was complete paralysis of both sides of the face.

All the symptoms detailed above appeared simultaneously. After three months, the paralysis of the face continued un-

changed. The patient remained completely deaf in the left ear, while the hearing of the right ear was somewhat improved. The sense of taste was at no time affected. When the patient was last seen, it seemed that the condition detailed at the last examination would be permanent.

Although there was no X-ray confirmation, the diagnosis of basal fracture could not be questioned. The injury to the facial nerves must have been above the geniculate ganglion, from the undisturbed taste function. It is probable that, from the involvement of the other cranial nerves, the tear was above the entrance of the nerves into the internal auditory meatus of each side.

CASE 2. (Oppenheim and Hallez). P. J., aet. 28, infantryman, was injured October 27th, 1916, by the bursting of a shell in close proximity. There was immediately loss of consciousness followed by prolonged anorexia. He could furnish no details as to how he was hurt. Consciousness returned sixteen hours later. The primary reports state that there were no external lesions. Double facial paralysis, bleeding from both ears, profuse nose bleed, and slight mydriasis on the left side were all apparent. After a day or two he became semiconscious again, and when he recovered complained greatly of nausea and vertigo. Epistaxis and bleeding from one ear returned again. He entered the otologic service two weeks after his injury for "concussion of the labyrinth, injury to the tympana, with infection on both sides and facial diplegia."

The face presented the appearance of a blank mask, without any expression, the creases of the forehead and of the face having completely disappeared, and there was slight edema of the face in both cheeks. There was no diplopia and visual acuity was normal. Speech was a little slow, scanning and especially embarrassed by pronunciation of the labials. Deglutition was normal.

It is interesting to note, that after the first visit of the patient's wife, in consequence of a violent emotion, the man presented a complete mutism for four days.

The cheeks were flabby, and mastication was considerably embarrassed because foodstuffs fell between the gums and the cheeks.

The soft palate was lifted normally, and there was no deflection of the uvula. The sensibility of the tongue was conserved, but the sense of taste was abolished in its anterior two-thirds.

The electrical examination made in January, two months after the injury, showed a degeneration reaction of the seventh pair, a little more marked on the right than on the left side.

The hearing on the right side was diminished, and there was suppuration of the middle ear. On the left side, the hearing was quite gone, and there was suppuration of the middle ear. The facial and auditory nerves seem therefore to have been involved, but no others were in any way injured.

The lumbar puncture, made in January, gave a clear lymphoid fluid with a little more albumin than normal, and a scarcely appreciable increase of the cellular contents.

The lesion doubtless, therefore, was in the Fallopian canal on each side. After several months sojourn in the service, the patient improved considerably, the eyelids could be partly closed and the forehead could be slightly furrowed. As to the course of the paralysis, the authors are in doubt. It might have resulted from a fracture through the petrous portions of both temporal bones, or it may have resulted from an immediate intratubal concussion. It is also possible that the injured man was thrown backward and sustained a fracture of the base through both temporal bones. In favor of this hypothesis are the nose bleed, the repeated bleedings from the ears and the prolonged coma. In support of the possibility of the shock of the explosion alone causing the damage in the petrous portions of the temporal bones, without fracture, the authors allude to two men, who, without any trace of external injury or even of a fall, presented simply as a result of the concussion a total, unilateral facial palsy.

Case 3. (Chatelin and Patrikos). The case when presented was of six weeks duration. There was a total, double paralysis of the peripheral type. On the left side the paralysis was absolute, while on the right side there was an indication of very slight movement in an effort to close the eye. The aspect of the face was characteristic. The forehead was entirely without wrinkles, the nasolabial folds had disappeared, the lips were half open and saliva drooled from the mouth. Speech was almost incomprehensible. Mastication was very difficult. Electrical examination showed complete degeneration reaction on both sides. The diplegia was the result of an accident. The wounded man was about to unload a heavily loaded wagon, when it was suddenly propelled backward and his head was caught transversely between the wagon and the wall. Released at once, the man bled from the left ear. The diplegia

manifested itself at once. For several days the face was very much swollen, and he had many ecchymoses in the temporomalar region. Eating was uncomfortable on account of the trismus. This, however, disappeared in a few days. The writer believes that the facial nerves were injured on both sides by a transverse fracture at the base of the skull, even if there were no actual symptoms of injured lesions of the organ of audition. It appears to the writer, that in the absence of the loss of taste and of the radiographic evidence of fracture of the base in this case, it is more likely that underneath the ecchymoses described in the temporomalar region, the facial nerves had been injured by compression after their exit from the stylomastoid foramina.

Case 4. (Marchand). Soldier, aet. 22, was thrown from an automobile truck, August 22nd, 1917. He had no recollection of how the accident happened. There was immediate bleeding from the ears, with epistaxis and bilateral facial paralysis. In the temporal region, there was a contused wound. The patient was unconscious for forty-eight hours. Diagnosis: Fracture of the base of the skull.

On the morning following admission, there was considerable palpebral and subconjunctival hematomata. There was also a hematoma at the nape of the neck. A lumbar puncture was made on the day of the accident, but in the original report no result of the examination is given. During six days the punctures were repeated.

He was admitted to the neurologic center at Bordeaux, on December 4th, 1917, when the facial paralysis continued. The record states that the movements of the face are nil on both sides. The patient cannot laugh, cry or whistle. Speech is difficult on account of the inability to pronounce the letters P. B. and M. Swallowing is normal, but to drink the patient must hold his head backward to prevent liquids from running out of his mouth. The sense of touch and pain is normal, both in the tongue and in the face. Salt, acids or sweets are not felt on the anterior two-thirds of the tongue on the right side, but the taste for bitter things is normal on both sides. In March, 1918, there was some voluntary movement of the lower portion of the left side, and the left eye could be completely closed. There was still a loss of the gustatory sense of the left side. On the right side, the paralysis presented improvement.

Case 5. (Ransohoff's own). Miss E. K., aet. 22, was injured Jan. 4th, 1919, in a street car accident. She bled from both ears, and was unconscious when admitted to the Cincinnati General Hospital. She was in coma for one hour. Vomiting followed. Then two general convulsions followed, lasting for five minutes. Patient suffered from severe headaches with pain in the ear. She had nausea for ten days. The pulse and temperature were normal. Complete left side paralysis was present, with slight ecchymoses over both mastoids.

On the morning of the third day after admission, the right side of the face became completely paralyzed. She was mentally sluggish. The right pupil was larger than the left. The left external rectus muscle was weak. The other eye muscles were normal. The patient was unable to close either eye, or to move the wings of the nose or her lips. The face was mask like. The lips flapped loosely on expiration. Speech was very indistinct.

Dr. Iglauer examined the ears. The right ear showed slight tinnitus. The drums were intact. There was no spontaneous nystagmus. Weber was lateralized to the left. Bone conduction was normal. The whispered voice was heard at eight feet. No hyperacusis was present. The eighth nerves are intact.

She can protrude her tongue in the midline and move it from side to side. The uvula hangs in the midline. The sense of touch is normal. The sense of taste is absent on both sides of the tongue, in its anterior aspect, two-thirds. The patient cannot distinguish sweets, vinegar or bitter things.

The X-ray shows an oblique fracture through the cranial base, running through the left mastoid process into the petrous portion of the temporal bone.

Paralysis disappeared from the right side of the face in two or three weeks, and during the next two months it disappeared entirely from the left side. Muscular control began in the lower portion of the face. The eyelids recovered last. Both sides recovered alike.

The lesion in Ransohoff's case was in each Fallopian canal, below the geniculate ganglion, and at a point above where the chorda tympani is given off. It was also below the point where the nerve to the stapedius is given off, since there was no record of hyperacusis.

Ransohoff refers to the theory of Moure, that the facial nerve in the Fallopian canal is composed of two sets of fibers:

one peripheral or superficial, which supplies the lower facial group of muscles, and the central or deep, which innervates the upper muscular group. But there has never been any anatomic demonstration of his theory, and the case of Ransohoff denies such an hypothesis.

Case 6. (Sachs). Patient, aet. 16, admitted October 11th, 1912, after a motorcycle accident. He was bleeding from the right ear and nose. No fracture could be made out by palpation. K.K. was negative. There was no ankle clonus and no Babinski reflex present. Left pupil was somewhat dilated. It did not respond to light. The left eye shows slight exophthalmos. Pulse was 138. Blood pressure 135 mm. The vessels of the eyegrounds were tortuous. Lumbar puncture could not be made. The right external rectus was completely paralyzed, and the left one slightly so. He could not pucker his lips or whistle. Neither facial nerve reacted to faradism. The strongest possible currents produced contraction, however. Quinin was not tasted in the anterior portion of the tongue, but he did recognize it on the posterior portion. Sugar and salt were recognized. When asleep both eyes closed, but when awake he could not close them. X-ray plates did not show basal fracture. On discharge, October 11th, it was noted that for the first time he was beginning to gain the use of the muscles about the mouth on the left side. This completes the cases referred to in Ransohoff's paper.

Case 7. (Levy and Tupper). Colored soldier, aet. 24, hurt June 9th, 1919. His head was caught between two railroad cars. He was unconscious for three weeks. On August 23rd, 1919, he entered Cook County Hospital, Chicago, Ill. Examination revealed excellent mental and physical condition. No Romberg was present. There were no abnormalities in gait, or in muscular, bone or genitourinary systems. No headache was present, or dizziness, or gastrointestinal derangement. He could not pronounce labials.

The face was expressionless, and the nasolabial folds were obliterated. The corners of the mouth hung down, especially the left. He could not wrinkle the forehead, pucker the lips, close the eyes, or show his teeth. To drink or smoke, he must roll up the lower lip and support it. The tongue was very dry and corrugated, but it could be protruded and moved in every direction. The uvula and soft palate were normal. The left eye showed marked paralysis of external movement. There was no nystagmus.

The sensation of taste for sour, sweet and salt was lost over the anterior two-thirds of the tongue. The sensibility of the face and tongue was normal. All reflexes were normal save the corneal and conjunctival. Electrical examination showed degeneration of both seventh nerves and of the muscles supplied by them.

X-ray examination (Dr. Blaine) showed a shadow indicating fracture into the middle fossa of the skull, close to the base and involving the right parietotemporal region.

Nystagmus tests:—Rotation to the right produced no nystagmus. To the left, six seconds of nystagmus were produced *but to the right*. There was no past pointing after rotation to the left. But after rotation to the right, the patient past pointed two inches to the right, with both hands. Hearing was fairly good. On October 9th, 1919, the right ear was douched with cold water, with no reactions from either horizontal or vertical canals. There was no past pointing, vertigo or nystagmus. Douching the left ear produced no response from the vertical canals. The horizontal canals gave slow nystagmus of fifteen seconds, and past pointing to the left. On October 13th, 1919, the vertical canals were stimulated by rotation, and the reactions were normal but shortened. Stimulation of the horizontal canals, by rotation to the right, gave nystagmus to the left, lasting for three seconds, and momentary past pointing with the left, but not with the right hand. Hearing was normal in the left ear, but somewhat diminished in the right ear. Bone conduction was normal on the left side, but prolonged on the right side of the head. Conclusion:—"The lesion of the vestibular apparatus is not due to a labyrinth destruction, is not peripheral."

Course:—The right facial nerve, especially the lower branch, showed marked and progressive improvement, but the left facial nerve and the left abducens remained stationary at the time of the last examination.

Summary and Discussion:—(1) There was bilateral degenerative facial paralysis of the peripheral type. (2) There was paralysis of the left abducens. (3) The chorda tympani was involved. (4) On the right side the hearing was impaired. (5) The lesion was one of the central portion of the vestibular apparatus.

The chorda tympani speaks for involvement of both facial nerves in their Fallopiian canals, i. e. in the petrous portions of the temporal bones. But bilateral fracture of the pyramid

bone is usually fatal (Marchand), and could not explain the involvement of the sixth nerve. We look for several or multiple lesions, which are the rule in severe concussions of the brain. (Jacob).

Case 8. (Author's). Oscar Gustafson, Swede, aet. 34, was caught in a cavein, while laying sewer pipe in a deep trench. He was completely buried, and when dug out was unconscious and bleeding from the ears and nose.



Fig. 1. Oscar G. Facial-diplegia. Appearance on admission to hospital. Face mask like.

He regained consciousness in a few hours, and it was observed that his face was completely paralyzed. Complete internal squint of the left eye was present, and partial of the right eye; he had diplopia. The record shows that he was very restless, complaining of severe pain in the head. The upper portion of the right chest was painful. At eight at night of the same day he was given 1500 units of antitetanic serum. (Figs. 1, 2, 3, 4, 5.)

His past history reveals nothing of the diseases of childhood save measles. He had influenza two years before. Otherwise, he has always been healthy. There is no history of lues, and the Wassermann tests were negative. Neither tuberculosis, cancer nor insanity affected any member of his family, past or present. His father died at the age of forty of pneumonia. His mother is living and well at the age of 77.

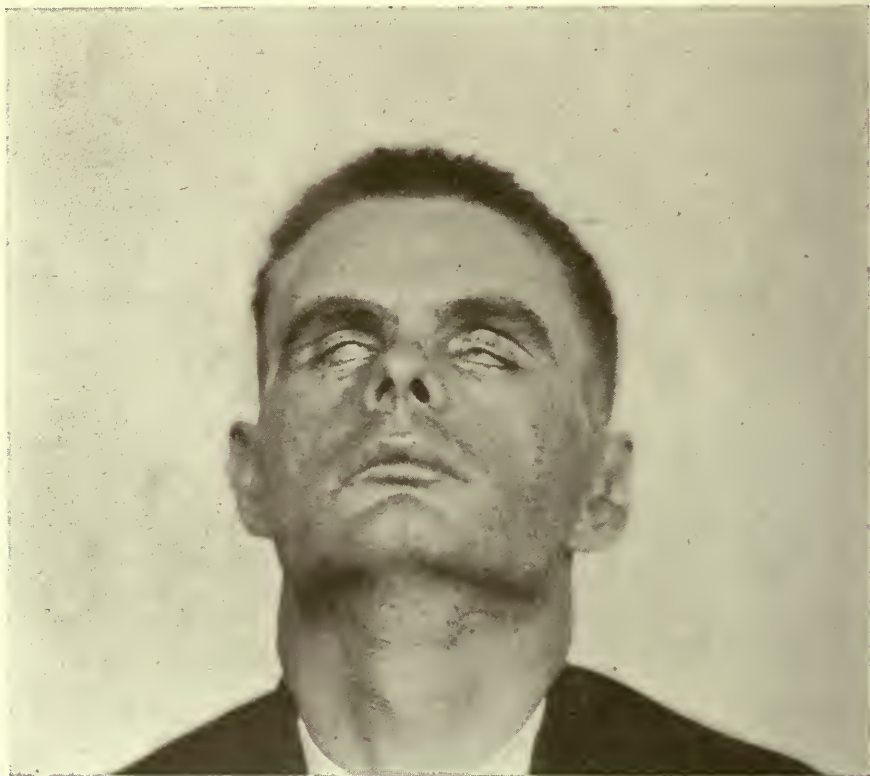


Fig. 2. Oscar G. Facial diplegia. Was unable to close the eyes.

One brother is living and well at the age of forty. Two sisters are living and well at the ages of 45 and 50 respectively.

Examination of his person:—The right clavicle was fractured. Below the right ear was a perforating wound with partial separation of the auricle from the head. Bloody spinal fluid was obtained at a pressure of 150 mm.

The face was expressionless. He could not show his teeth or whistle. He could not close his eyes. His speech was very indistinct because of the lack of lip action. Internal strabis-

mus was very marked in both eyes. His tongue and lips were quite dry. He had trouble eating and drinking, because the food and drink would drop in between the cheeks and jaws.

Sensation to touch and pain remained unimpaired. The thorax expands fairly well, and the breath sounds are of good quality and there are no rales or rales. The left border of the heart is well inside the nipple line. No murmurs, shocks or



Fig. 3. Oscar G. Facial diplegia. Marked paralytic squint of the left eye. Abducens paralysis. Partial internal squint of the right eye.

thrills are present. The abdomen was slightly tender above the symphysis, no distension is present, and no tumor masses can be made out. The genitourinary apparatus was normal. The skin is clear. The joints are flexible. The costochondral end of the right fourth rib was depressed and pressure caused pain. There is no adenopathy. The biceps, knee jerks and other reflexes are normal. The pupils are equal and respond fairly well to light and shade. X-ray examination re-

vealed a dislocation of the atlas on the axis. Through the pedicle of the fourth cervical vertebra is an oblique fracture, and a fracture of its spinous process. While the X-ray plate is not satisfactory, yet it seems to show a fracture of the base of the skull passing through both temporal bones. (Fig. 6.)

The soft palate and uvula are normal. He can protrude his tongue and move it in every direction.



Fig. 4. Oscar G. Facial diplegia. Unable to rotate the left eye outwards.

His temperature and pulse was as follows:

July 5th. Temperature: 100.4 degrees; Pulse: 64.

July 17th. Temperature: 97.6 degrees; Pulse: 50.

July 19th. Temperature: 98. degrees; Pulse: 48.

Otherwise both ran a normal course. Both ears discharged pus freely.

On July 21st, on account of the eye condition, I was asked to take charge of the case by Dr. A. C. Arnett, his physician.

All the above conditions were present on that date, and in addition it was observed that the upper lid of the left eye drooped markedly. Both eyes had marked limitation of motion temporally.

Hearing tests were made. Weber was not lateralized. Rinne was negative: Right ear: Bone conduction 20 seconds, and air conduction, 14 seconds. Left ear: Bone conduction 25 seconds, and air conduction 22 seconds. The tuning fork



Fig. 5. Oscar G. Imperfect rotation of the right eye outward.

has 128 double vibrations per second and is weighted to guard against overtones. A stop watch was used as a timer. Both membranæ tympanorum were ruptured, and pus was present in the lips of the wounds.

The pupils were normal and responded well to light and shade. The fundi appeared normal. Protecting glasses with wire mesh temple guards were ordered to protect the eyes against extraneous substances, of which the paralyzed eyelids could not dispose.

On July 21st, 1921, tests of sensation were made with the esthesiometer, with the following results:

Region:	Right	Left
Supraorbital	6 mm.	9 mm.
Infraorbital	6 mm.	16 mm.
Upper lip	4 mm.	5½mm.
Mental	7 mm.	10 mm.



Fig. 6. X-ray picture to show the dislocation of the atlas upon the axis. Also it shows the fracture of the pedicle of the fourth cervical vertebra. Taken shortly after the accident.

The sense of smell appeared unimpaired. The sense of taste was markedly impaired. He did not, and does not yet, recognize either quinin or vinegar placed anywhere upon the tongue. Sugar and salt are barely recognized on the posterior portion of the tongue, but not on its anterior portion.

On August 3rd, he was allowed out of bed.

September 15th, we made record of his hearing as follows:

Weber not lateralized. Rinne, right ear; bone conduction 25 seconds; air conduction 32 seconds. Left ear; bone conduction 32 seconds; air conduction 28 seconds. Range of hearing 128 d. v. s. to 38,000 d. v. s.

For the sake of comparison I will here insert the record of the last tests made, June 26th, 1922:

Weber is now lateralized to the right. Rinne, right ear; bone conduction 22 seconds; air conduction 51½ seconds. Left ear: bone conduction 23 seconds; air conduction 20 seconds. Low tones 128 d. v. s. The upper limit is 40,000 d. v. s. Both ear drums are healed over.

Turning tests resulted thus: To the right ten times, nystagmus twenty seconds and of good amplitude. To the left ten times, nystagmus nineteen seconds, and of good amplitude. Falling tests are normal. He past points normally with both arms. No douching tests were made because of the condition or his ear drums. There were no fistula symptoms.

On September 15th, 1921, the tests for sensation were repeated with the following result:

Region:	Right	Left
Supraorbital foramen	8 mm.	10½ mm.
Infraorbital foramen	6½ mm.	9½ mm.
Mental foramen	5 mm.	4 mm.
Inframaxillary	14 mm.	18 mm.
Preauricular	11 mm.	12 mm.

The last tests thus were made August 3rd, 1922, as follows:

Region:	Right	Left
Supraorbital foramen	19 mm.	10½ mm.
Infraorbital foramen	9 mm.	5½ mm.
Mental foramen	9 mm.	8 mm.
Tip of tongue	3 mm.	

The left eye gave us a great deal of trouble. Imperfect closure of the eyelids produced exposure keratitis. Iritis began to show itself August 18th, 1921, with ulceration of the cornea, just below its center. Atropin and bandage with hot applications controlled the pathologic condition pretty well.

Lugol's solution of iodine was also applied locally. By September 11th, the healing was complete, with the usual scar. The ulcer recurred October 14th, and although the same treatment was pursued, yet it progressed, and on October 26th, we were compelled to raise a Kuhnt flap to prevent perforation of the cornea. The stitches pulled out on the 27th, but the ulcer had practically healed over. October 29th, the cornea threatened perforation and Descemet's membrane only was intact. So another Kuhnt flap was raised and the ulcer covered. On November 2nd, the stitches pulled out, but by the 7th, the healing was practically complete.

Prince's method of pasteurization was tried without avail, as well as the thermophore of Shahan, likewise without result.

Inasmuch as others had made zinc-fluorescein and mercury-fluorescein (mercurochrome), not to be outdone I proceeded to make silver-fluorescein, by mixing a two percent solution of silver nitrate with an equal quantity of the usual fluorescein solution used routinely in our offices to outline corneal ulcers. This we used to combat intercurrent suppuration, but it availed naught.

On December 23rd, the ulcer recurred with rapid thinning of the corneal substance. We raised another Kuhnt flap from below, again, and covered the ulcer. It was effective in preventing perforation. But the ulcer did not heal in well this time, and again on January 4th, 1922, we raised another Kuhnt flap, because the ulcer threatened perforation. This was the last one needed, for the eye cleared promptly and the inflammation left it completely. Of course, there is a large scar extending up into the pupillary area, about one-half being occupied thus. Blood vessels can be seen coursing in from the limbus upward, nature's way of healing the ulcer.

Recession of the eyeballs into the orbit was noted in due time, and on November 29th, 1921, I made measurements with the exophthalmometer of Hertel. The right eye measured 12 millimeters, and the left 11 millimeters. I checked this result with two normal individuals. One was the male attendant on the floor of St. Elizabeth Hospital, where our patient was confined, and the instrument measures 20 millimeters for the right eye, and 18 millimeters for the left eye. Sister A's eyes showed 18 millimeters for each eye. At the present time, the exophthalmometer measures 11 millimeters for each eyeball.

The vision for the right eye, June 26th, 1922, is 20/20,

which is increased to 20/15 with a plus .50 D. cylinder, axis 90 degrees. The vision of the left eye is 20/50 and no lens helps.

The blood examination was and is normal, and so is the urine test.

The bacteria present in the conjunctival sac were the



Fig. 7. Taken September 1, 1922. The dislocation still persists. The separation of the temporal and parietal bones shows. The fracture of the petrous portions of the temporal bones shows in the X-ray plate but cannot be made to show in the photograph.

staphylococcus pyogenes albus and aureus, and the micrococcus catarrhalis.

He is gradually improving, and at the present time his face is more mobile. He can now close his eyes pretty well. His speech is more distinct, as he has gained some control over his lips.

This patient was treated by an osteopath while under my care. That there may be no vainglorying, I had X-ray plates made September 1st, 1922. The dislocation of the atlas upon the axis still persists. Furthermore, a distinct line of separation of the parietal and occipital bones is apparent. The line of fracture into the petrous portion of the temporal bone still shows. (Fig. 7.)

Observations in general as to this patient:—

(1) What is the meaning of the hemorrhage from the nose and ears?

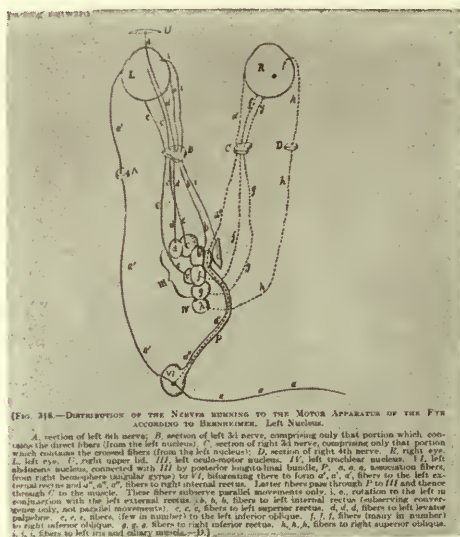


Fig. 8. Traumatic facial diplegia, showing origin of fibers going to the levator palpebrae superioris.

(2) What is the meaning of the ptosis of the left upper eyelid?

(3) What is the meaning of the loss of hearing and its recovery in part?

(4) What is the meaning of the double facial paralysis?

(5) What is the meaning of the abducens paralysis?

(6) What is the meaning of the reduced unilateral sensation?

(7) What is the meaning of the apparent recession of the eyeballs into the orbit?

(1) As he has survived, detailed examination of the cranium and its contents is impossible. But he suffered a fracture

of the base of the skull involving, no doubt, the petrous portion of the temporal bones, and probably the lesser wing of the sphenoid. The skull must have endured intense lateral pressure.

(2) The ptosis of the upper lid of the left eye is hard to explain, because the section of the nucleus of the nerve supplying the eyelid lies so far in advance of the other divisions of the nerve, as the lantern slide will show. Yet the median longitudinal bundle connects all the nerves involved. (Fig. 8.)

(3) As the static portion of the labyrinth was not involved according to the tests applied, the cochlear portion was dam-

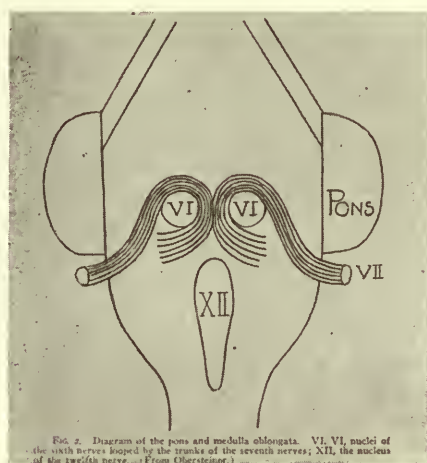


Fig. 9. How the fibers of the seventh nerve encircle the nucleus of the sixth cranial nerve.

aged somewhere between the internal auditory meatus and the origin of the nerves. However, this is pure speculation. The vestibular tests indicate that there was no involvement of the pons.

(4) The double facial paralysis may be explained in a number of ways. It is probable that the basal fracture may have compressed both nerves in the Fallopian canals. Or the compressing hemorrhage may have passed over from one nerve to the other, as the lantern slide will show. Again the hemorrhage may have been in the nuclei of origin.

(5) The involvement of the abducens nerves is readily demonstrated by the lantern slide. It will be observed that the nucleus of the sixth nerve lies under the origin of the seventh

nerve. Moreover, the fibers of the seventh nerve wind around the nucleus of the sixth nerve in the floor of the fourth ventricle. This tends to prove that the origin of all his trouble thus is a hemorrhage into the nuclei of origin. (Figs. 9, 10, 11.)

(6) The reduced unilateral sensation all over the left side of the face may be explained by a consideration of the anatomy of the fifth nerve, and I quote liberally from Deaver's *Surgical Anatomy of the Head and Neck*. "The fifth cranial nerve, trigeminus or trifacial, the largest of the cranial nerves, arises

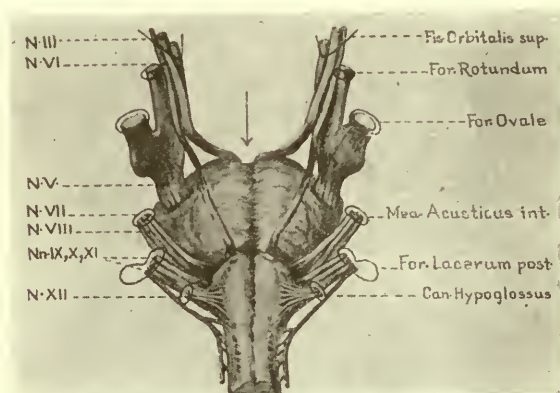


Fig. 10. Facial diplegia (traumatic). From Cushing's "Tumors of the Acoustic." The relation of the cranial nerves to each other after emergence from their nuclei.

from the sides of the pons by two roots, a large posterior or sensory root, and a smaller anterior or motor root. These roots can be traced to the floor of the fourth ventricle and to the gray matter in the lower part of the medulla oblongata, and in the upper part of the spinal cord. It is the only cranial nerve which resembles a spinal nerve in arising from two roots. The trifacial is a mixed nerve. It distributes sensory filaments to the dura mater, pia mater, orbit, eyelids, nose, gums, teeth, tonsils, palate, sphenoid cells, ethmoid cells, frontal sinus, maxillary sinus, nasal fossae, pharynx, articulation of the lower jaw, ear, parotid gland, scalp, forehead, and face, and gustatory filaments to the anterior two-thirds of the tongue." I may state, however, that the distribution to the tongue is otherwise described by other anatomists. According to Cunningham, the anterior two-thirds of the tongue is supplied by the chorda tympani, so far as the taste buds are

concerned. The posterior third is supplied by the glosso-pharyngeal. The epiglottideal taste buds are supplied by the inferior laryngeal nerve. For general sensation the tongue relies upon the lingual branch of the mandibular division of the 5th nerve. The same hemorrhage into the nuclei of origin of the other nerves involved also spread to involve the origin of the left fifth nerve, damaging also his sense of taste.

(7) The recession of the eyeballs into the orbit may be explained as due to a trophic change going on in the orbit, probably absorbing some of the fat therein.

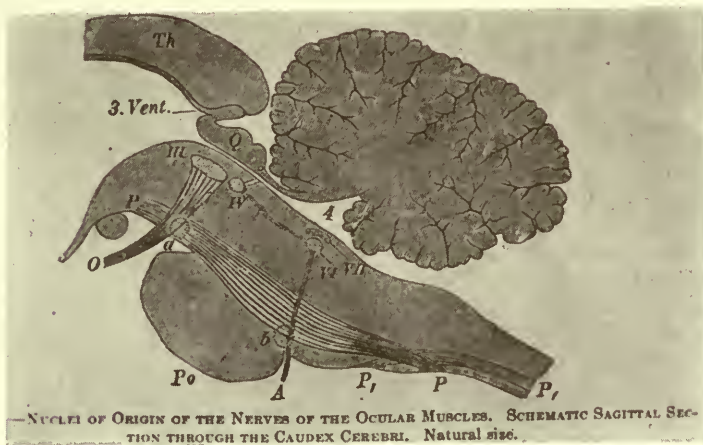


Fig. 11. Traumatic facial diplegia, showing origin of the nerve in the floor of the fourth ventricle

ULTIMATE CONCLUSION.

Because postmortem examination is impossible, we cannot determine the exact lesion and its location. But when all observations are put together, we are almost forced to the conclusion that the nuclei themselves in the floor of the fourth ventricle must have been affected by hemorrhage, which, as it is absorbing, is bringing recovery to the parts affected.

A remarkable feature of all the cases of this trouble reported to date is that not one perished. Hence we are driven to the conclusion that the prognosis is reasonably good for improvement at least.

OUR COMPENSATION LAWS.

This patient revealed to me some phases of our compensation laws not noted heretofore. As a rule, the laws of the var-

ious state provide a maximum of sixty days' hospital care and physicians' services. Patients injured thus cannot recover within the period of sixty days. Hence the insurance company can hide behind this phase of the law and refuse to pay either hospital or doctor for more than sixty days' services. Such patients are laborers on comparatively small pay. As matters now stand, the doctor is compelled to stand for what the insurance company should pay. Let us go to our homes in the various states to see that the compensations laws are so amended, that these companies assume the entire cost of medical and hospital care for these unfortunates, for their pay goes on in a measure, until recovery is acquired. We should receive our just dues.

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TRANSACTIONS
OF THE
TWENTY-SEVENTH ANNUAL MEETING
OF THE
American
Academy of Ophthalmology
and Oto-Laryngology

OTO-LARYNGOLOGIC DIVISION

THE EARLY DIAGNOSIS OF LARYNGEAL TUBERCULOSIS.

FRANK R. SPENCER, A.B., M.D.

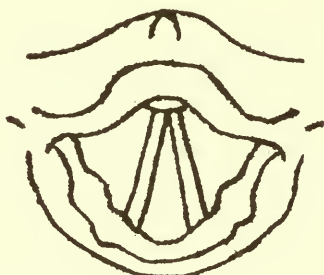
BOULDER, COLORADO.

The early diagnosis of laryngeal tuberculosis is of so much importance, that it has seemed to the writer that it should be emphasized more than it is. Many authors, e.g., Minor¹, Curtis², Thompson³, Carmody⁴, Grayson⁵, and others, have laid stress upon the value of an early diagnosis. Brown⁶ states that laryngeal tuberculosis "occurs in about 25 per cent or more of adults with pulmonary tuberculosis, slightly more in men than in women, and next to tuberculous enteritis and colitis is the most frequent complication of pulmonary tuberculosis, due most likely to direct infection of the part with the sputum. Even early cases—cases in the incipient or minimal stage—are not spared (12 per cent), but as the pulmonary disease progresses the laryngeal complication becomes more frequent (moderately advanced, 26 per cent; far advanced, 45 per cent). The significance, then, of a complication so frequently seen among patients with pulmonary tuberculosis cannot be exaggerated." Other things being equal an early laryngeal diagnosis means a better prognosis, a shorter period of treatment, more complete restoration of the voice and sometimes an earlier pulmonary diagnosis; because some patients naturally may consult a laryngologist before they do an internist.

While the time allotted to one essayist will not permit a discussion of all the phases of this subject, I hope to emphasize some of the aspects which have impressed me during almost eighteen years' experience in the diagnosis and treatment of this disease. Levy⁷ has said, "No subject in medicine possesses a more extensive or interesting history than tuberculosis, nor can any disease boast of more attempts at solving the problem of its treatment with less permanent, definite and satisfactory results." It is because of this situation that I am particularly interested in the better prognosis which an early diagnosis affords. Thompson³ states that "progress is ready to hand in the making of an earlier diagnosis of local infection" in speaking of laryngeal tuberculosis.

Freudenthal⁸ believes "the prognosis of an established tuberculosis of the upper air passages is better than it used to be years ago." He also states that "all patients suffering from pulmonary tuberculosis should be advised to undergo a laryngologic examination, not only when the disease is diagnosed or upon their entering a sanitarium, but also at regular intervals, irrespective of their complaints." Mullin⁹ has expressed a similar opinion regarding laryngologic responsibility.

One of the very first things of which a patient will complain, in the early involvement of the larynx, is a slight huskiness of the voice. Usually, this is at first noticeable after getting up in the morning, and is often relieved by coughing or clearing the throat. By such act, in the very early stage, the patient may be entirely freed of this slight hoarseness for the rest of the day. If the patient becomes tired, the hoarseness is more apt to return. Often, on the other hand, for a period



Normal Larynx.

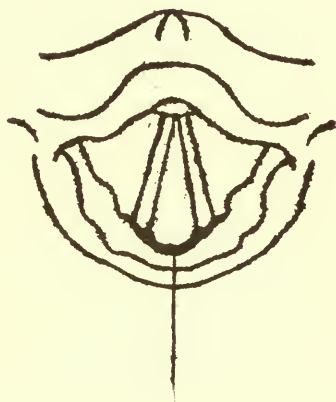
Fig. 1.

of weeks or months he is free from hoarseness upon arising, After a time, if the disease progresses, there may be slight hoarseness throughout the day, which is more noticeable if the patient uses his voice for any length of time in conversing with friends. Talking is, of course, not encouraged after the patient has been examined by a laryngologist.

Solenberger¹⁰ found "mere weakening of the voice, vocal fatigue on talking, hoarseness, a sense of dryness in the throat, troublesome localized tickling, a lump like tickling on swallowing, pain, a feeling of languor or debility and increased dry cough," all important in the early stage of laryngeal disease. He also mentions recurrent acute laryngitis as a frequent symptom. He states that "these signs no doubt often

herald the approach of the disease, and should always be an indication for speedy laryngoscopic examination. But they are also signs of actual involvement. Sometimes, nearly all these external signs are absent in the process of involvement even to the point of disintegration."

Four main types present manifestations worthy of our consideration clinically, when we examine the larynx. We may find any one or all four of the following in any case:



Infiltration.

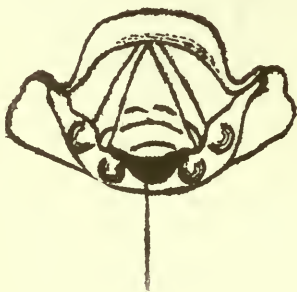
Fig. 2.

1. Infiltration.
2. Tuberculoma.
3. Ulceration.
 - a. Acute or active.
 - b. Chronic or sluggish.
 - c. Superficial.
 - d. Deep.
4. Edema.

Fetterolf¹¹ has classified the ulcerative stages as follows: (a) Infiltration with superficial ulceration, (b) infiltration and deep ulceration, and (c) infiltration with superficial and deep ulceration. This classification has much in its favor, because we know that frequently the ulcers form on an infiltrated base.

Infiltration, in our experience, has been one of the very earliest symptoms, and has usually become manifest first over the posterior laryngeal wall, the interarytenoid sulcus or the

arytenoids. Less frequently, the epiglottis or the ventricular bands are first involved. If the infiltration takes place slowly (and it usually does) the mucous membrane looks thick and has the dark red appearance so often seen with hypertrophy of the inferior turbinate. This type is sometimes designated as hypertrophic tuberculous laryngitis. In addition, the membrane looks stretched. It is not unlikely that many patients recover spontaneously in this very early stage, just as we know they recover from slight pulmonary lesions. Mullin has reported, before the Colorado Oto-Laryngological Society, the finding of small calcified areas in the larynges of patients who formerly had pulmonary tuberculosis. He first demonstrated this with the aid of the X-ray while in the army.



Infiltration.

Fig. 3.

The following slides or drawings illustrate infiltration: Figs. 2, 3, 4, 5, 7, 11, 12, 13, 14, 15, 16, 18, 24, 25 and 26.

Trautmann¹² has stated that tuberculoma is among the earliest laryngeal manifestations, and some of the drawings illustrate this. Slides or figures, 4, 6, 12, 17, 25 and 28, show tuberculoma. It is tuberculoma in particular, and especially in the early stage, which is often mistaken for a papilloma.

The ulcers are quite small in the beginning and are quite superficial. The edges are irregular in outline and are often spoken of as "mouse-nibbled." The area of hyperemia about the ulcers is usually absent, but may be present later from rapid breaking down of a tubercle, due to mixed infection. A deep punched out ulcer with a red, regular border is suggestive of syphilis. Lues must often be excluded by the history,

by a general physical examination and by Wassermann or other laboratory tests. Hurd's¹³ paper gives valuable differential points in doubtful cases, such as the therapeutic test. Carmody⁴ states that "where there is much scar tissue we may and do frequently have syphilitic infection also." Ulcers are most frequently observed on the epiglottis, ventricular bands, aryepiglottic folds, true cords, interarytenoid sulcus, arytenoids and posterior laryngeal wall. An ulcer forms at the point where a tubercle has, by the pressure exerted, interfered sufficiently with the blood supply at the surface to produce superficial necrosis. The caseation within the tubercle is usually due to the toxins produced by the tubercle

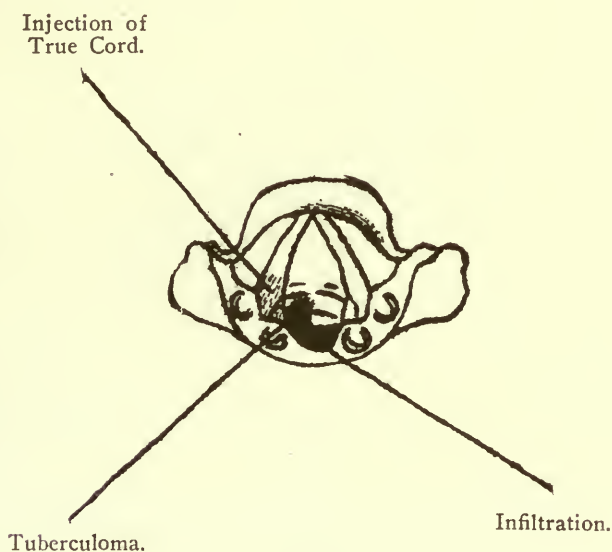


Fig. 4.

bacilli. Add to these two conditions a virulent mixed infection and the ulcerated area will break down rapidly with the result that the perichondrium may become involved. Rapid necrosis of the cartilages soon proves fatal. Lockard¹⁴ has shown, microscopically, that the necrosis of the cartilage of the epiglottis may be due entirely to tubercle bacilli and that it is not necessarily dependent upon a secondary mixed infection.

Acute ulcers often spread with great rapidity and are accompanied by a variable amount of pain. Chronic or sluggish

ulcers are far more frequently encountered than the acute or active. Since they are of a sluggish type pain is rather infrequent. Slides or drawings, numbers 8, 9, 10, 11, 12, 13, 14, 19, 20, 23, 24, 25, 26, 27 and 28, all illustrate ulcerations. Ulcers may also be classified as superficial and deep.

Edema of the larynx is very apt to become manifest at a later stage when the pressure is rather seriously interfering with the blood and lymph supply. This condition can be produced by a large number of small tubercles or by a few large ones, especially if the tubercles have increased in size rapidly. The edema produces the so-called typical "ashen-gray" ap-

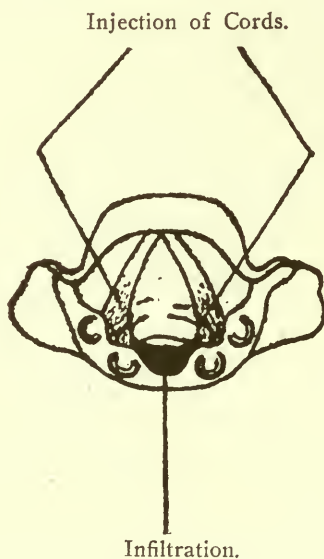


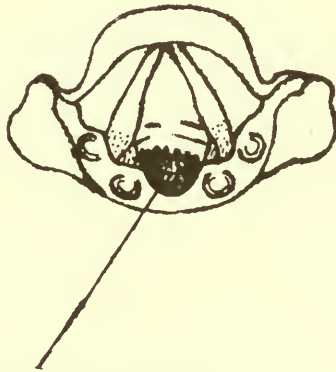
Fig. 5.

pearance and reminds one of a hyperplastic middle turbinate or a polyp. This has been spoken of formerly as "gray infiltration." Individual tubercles or groups of tubercles and ulcers are often obscured by this edema. Edema is illustrated by slides or drawings, numbers 16, 17, 21, 22, 23 and 27.

Frequently, at a very early stage, an examination of the larynx will reveal only a slight, dark red color of the entire laryngeal mucosa, with a loss of luster, and a slight pink tinge of the true cords. This slight pink tinge is usually confined to the posterior third of the true cords. However, very close inspection at this time will often show, as compared with the

appearance of the normal larynx, slight thickening of the mucous membrane over the posterior laryngeal wall. Ellis, in discussing Freudenthal's¹⁵ paper, urges an early diagnosis, because of its importance based upon such findings as the above.

While the above picture represents little more than a chronic catarrhal laryngitis, it should always arouse suspicion, especially in patients who have pulmonary tuberculosis of one or both lungs. So much involvement of the larynx may be produced entirely by the irritation from coughing. It may also be produced by the postnasal discharge from accessory sinus disease. While repeated physical examinations of the lungs at this time may fail to show any lesions, the stereo-



Tuberculoma Resembling Papilloma.

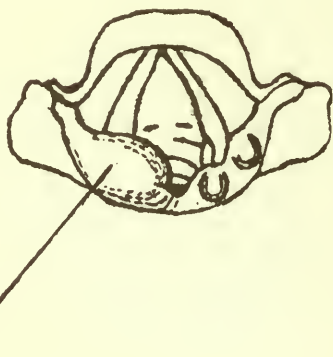
Fig. 6.

scopic X-ray films of the chest will usually reveal a tuberculous lesion of one or both apices, thus showing that the laryngeal lesion is secondary to the pulmonary disease. Dean¹⁶ and Mullin¹⁷ have shown the desirability of cooperation between the laryngologist and internist, if the best results are to be obtained for the patient. Bane¹⁸ states that "vigorous efforts should be put forth to check the local disease."

With all of the modern means at the disposal of the internist, an early diagnosis of pulmonary tuberculosis is usually not difficult or impossible. The valuable aid rendered by the roentgenologist should not be underestimated.

Though some cases have been reported in the literature as primary laryngeal tuberculosis, it is very doubtful if such

cases exist. The classification of primary laryngeal tuberculosis may be useful for teaching purposes, but is of very little practical value with the modern methods of diagnosis of pulmonary lesions now at our disposal. The cases are so extremely rare that they hardly deserve serious consideration. Many laryngologists of wide experience, such as Doctor Robert Levy of Denver, state that primary laryngeal tuberculosis has almost never been encountered in their many years of experience in the diagnosis and treatment of laryngeal tuberculosis. Hastings¹⁹ states that he has never seen a primary case. He also says "tuberculosis of the larynx is



Club Shaped Arytenoid.

Fig. 7.

rarely, if ever, primary. The primary focus may be undemonstrable except by autopsy. In view of the numerous positive tuberculin test, observations and confirmatory autopsy records in apparently nontuberculous patients a diagnosis of primary tuberculosis of the larynx can well be questioned. Certainly, a verdict of 'not proven' so far as its being primary is legitimate, until a searching autopsy is made." In discussing Hurd's¹³ paper, Levy made a similar statement.

I believe that some of the authors who have reported primary laryngeal tuberculosis have been misled by the fact that the subjective laryngeal symptoms were manifest before the diagnosis of the pulmonary disease had been made. Arrowsmith²⁰ states, "It is conceded today that primary laryngeal tuberculosis probably never occurs and that most individuals who exhibit tuberculous manifestations in mature life

have been infected in childhood or infancy, the infection remaining latent as a rule, until some adventitious stimulus spurs it into activity in any or all of its protean forms." Wood²¹ believes that "it is unquestionable that primary tuberculosis of the larynx has occurred, though it is exceedingly rare." Freudenthal⁸ believes "primary tuberculosis of the larynx does occur, and more frequently than has hitherto been

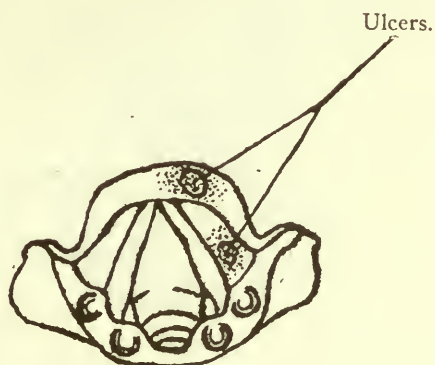


Fig. 8.

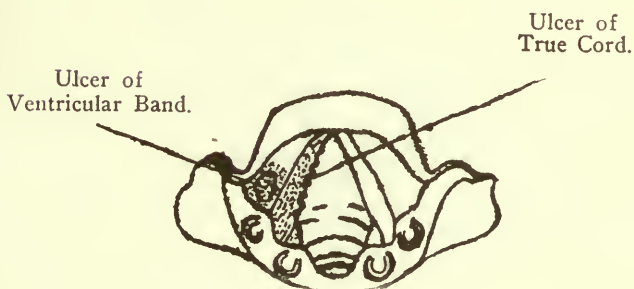


Fig. 9.

accepted." His long experience in the diagnosis and treatment of this disease, coupled with his mature judgment, certainly gives the weight of authority to his decision. Chappell²² held the same opinion.

That the larynx sometimes heals spontaneously in this very early stage there can be very little doubt. Levy²³ has recently mentioned this. Mullin, as previously stated, has found with the X-ray, calcified areas in the larynx in patients

who formerly had pulmonary tuberculosis, but who are not aware that they have ever had a tuberculous laryngitis. On the other hand, such cases are certainly more amenable to treatment than when diagnosed later. However, a few cases will not respond to treatment even with this early diagnosis, especially if the pulmonary lesions are steadily advancing.

V-Shaped
Ulcer of
Epiglottis.



Fig. 10.

Ulcer of False Cord. Ulcer of True Cord.



Infiltration.

Fig. 11.

If the infiltration over the posterior laryngeal wall increases beyond the slight thickening of the mucous membrane above mentioned, there can be very little doubt about the involvement being due to an infection with tubercle bacilli. The fact that the posterior laryngeal wall first shows clinical changes has been explained by the fact that sputum usually

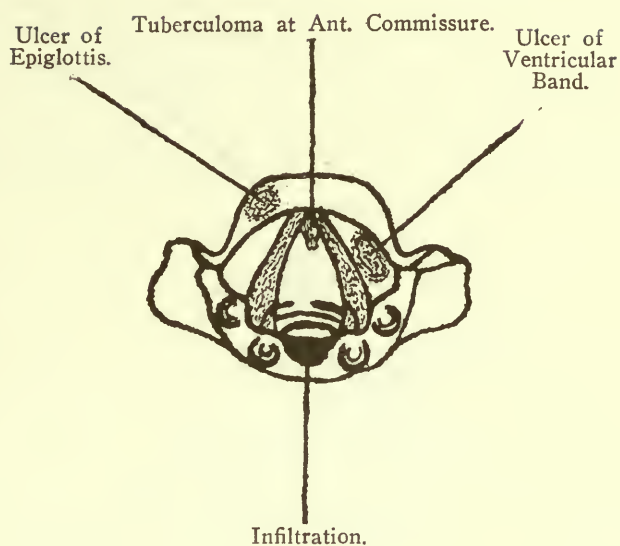


Fig. 12.

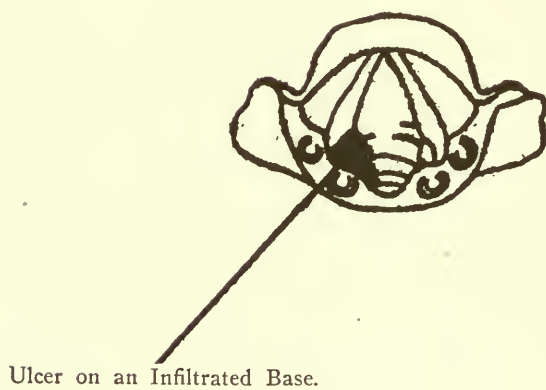


Fig. 13.

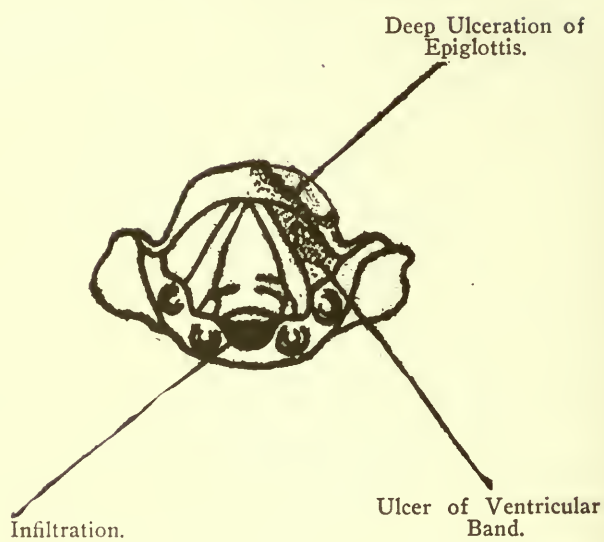


Fig. 14.

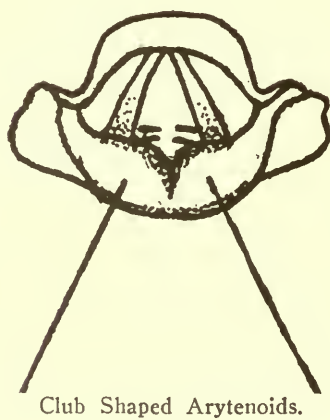


Fig. 15.

remains in contact with the posterior wall during sleep, instead of being expectorated as it is during the hours while the patient is awake. Minor²⁴ has recently shown many of the early manifestations involving the posterior half of the larynx. If the stage of infiltration is carried a step further, it is very apt to involve the arytenoids with the production of the so-called "club shaped" arytenoids. The club shaped appearance

Infiltration of Epiglottis.



Fig. 16.

Tuberculoma
of Epiglottis.

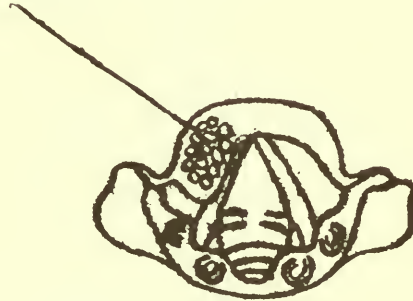


Fig. 17.

has often been termed pathognomonic of this disease. One must, of course, exclude syphilis before reaching such a conclusion, even in patients who have pulmonary tuberculosis, because it is not altogether unusual to find syphilis and tuberculosis in the same patient.

With the club shaped enlargement of the arytenoids, due to infiltration, the interarytenoid sulcus is also more or less obliterated. It is this obliteration of the interarytenoid sulcus

which has so often impressed me as an early sign. If the infiltration of the posterior laryngeal wall, with or without involvement of the arytenoids or interarytenoid sulcus, has taken place quite slowly, the larynx retains its dark red appearance and reminds one of the color of a hypertrophic inferior turbinate, because he does not see individual tubercles under the mucous membrane at any point macroscopically. On the other hand, if the infiltration takes place quite rapidly, due to the formation of either a single large tubercle or a series

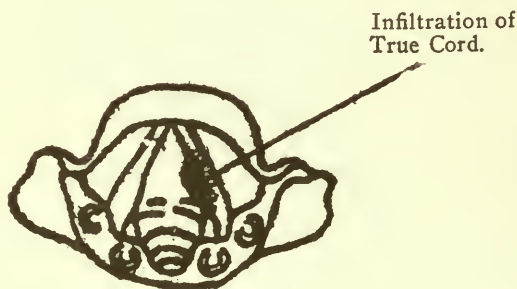


Fig. 18.

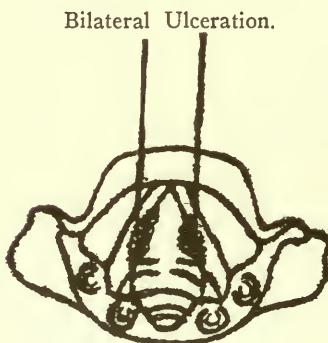


Fig. 19.

of small ones, the larynx becomes more or less edematous and has the so-called typical "ashen-gray" appearance. It has never seemed to me that the "ashen-gray" appearance is necessarily typical of laryngeal tuberculosis, except in the later stages of the disease, or in case the process is spreading rapidly. I usually feel when I see the ashen-gray color of the mucous membrane that the laryngitis has lasted much longer than the patient realizes. Lockard's²⁵ text-book illustrates this well. Lockard's²⁶ more recent article is well worth

Ulceration of False Cord and
Epiglottis.

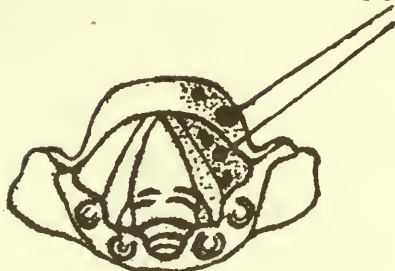


Fig. 20.

Marked Swelling of Epiglottis.

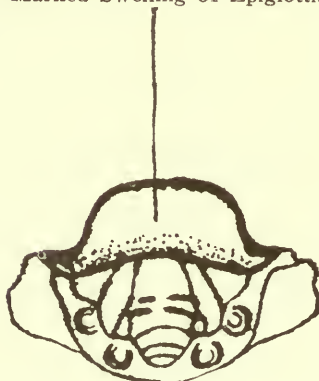


Fig. 21.

Marked Swelling of Epiglottis.



Club Shaped Arytenoids.

Fig. 22.

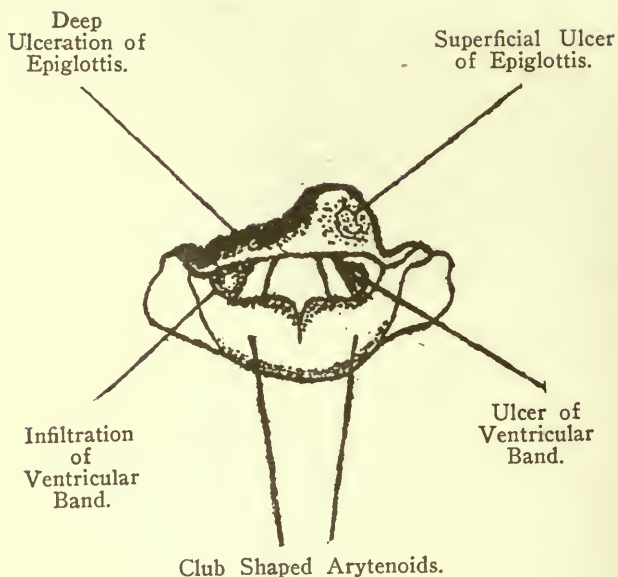


Fig. 23.

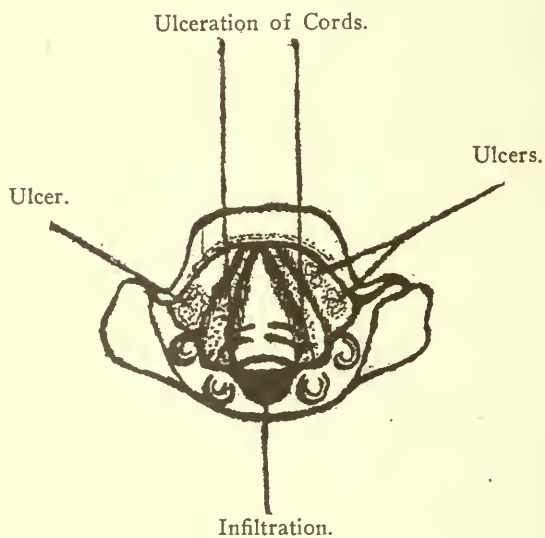


Fig. 24.

reading. Mullin⁹ has urged laryngologists not to wait until there are advanced and unmistakable evidences of laryngeal involvement before attempting to do something for the patient.

The examiner may find a small pyramidal swelling which projects out into the lumen of the larynx from the posterior wall, often before the swelling of the posterior wall attracts very much attention. These small pyramidal swellings are in the early stages so small that they must be examined for very carefully, otherwise they are overlooked in the mass picture of the larynx. They are readily obscured if the patient gags. The apices of these pyramidal swellings are often irregular and have a mouse-eaten appearance due to the

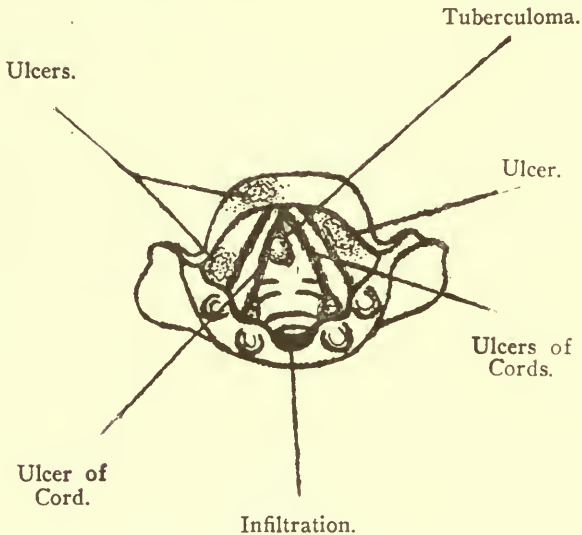


Fig. 25.

ulceration and loss of substance at the apex. The ulcer has a light gray coating of thin mucopus and the edges of the ulcer are irregular. These small ulcers are frequently found over the arytenoids and on the posterior surface of the epiglottis. They may vary greatly in size from one or two millimeters to several millimeters. In other patients, without any microscopic evidence of infiltration of the arytenoids, there will be some infiltration of the cushion of the epiglottis.

While we know from experience that the posterior laryngeal wall is more frequently involved than the anterior wall, we must not overlook the fact that early infiltration at the base of the epiglottis is of diagnostic importance in the early

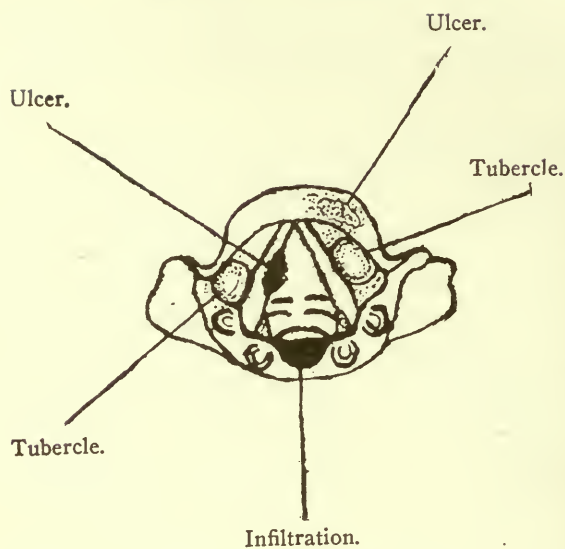


Fig. 26.

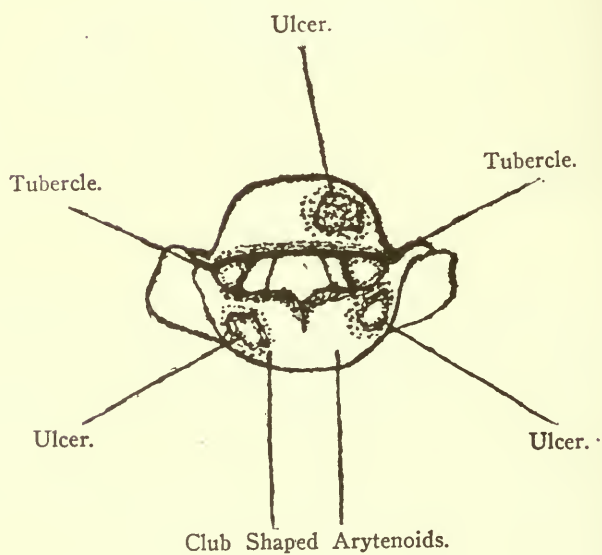


Fig. 27.

recognition of laryngeal tuberculosis. Here again we must be on the alert for a syphilitic infiltration, especially if ulcers are present. The fact that syphilitic ulcers are usually deep and that they have a punched out appearance, with very dark red borders, in contrast with the superficial mouse-eaten ulcers of tuberculosis, helps in the differentiation. If we add to any one of the above pictures infiltration of the false cords, which is apt to involve the posterior third first, we are dealing with some of the later manifestations of laryngeal tuberculosis which presents a more or less unmistakable picture, and the prognosis and treatment of these are not so encouraging.

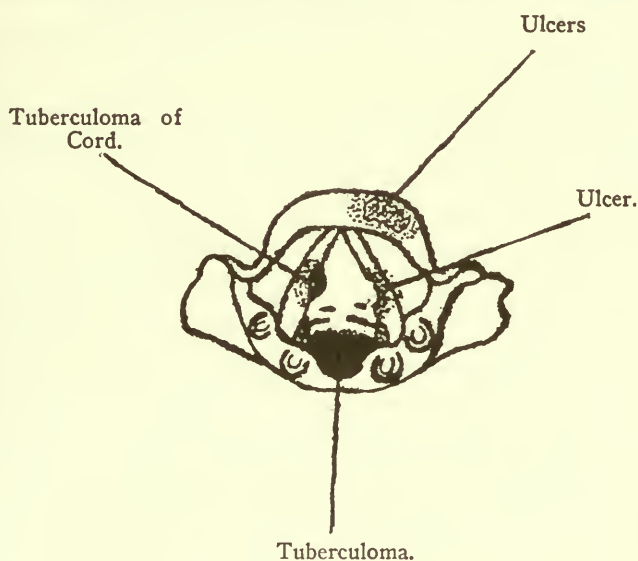


Fig. 28.

Coakley²⁷ states that laryngeal tuberculosis is most frequent between the ages of 20 and 45 and this corresponds with our experience. It is a disease which strikes the hardest in the prime of life and during the period of greatest usefulness of the individual. Thomson²⁸ states that "tuberculosis is one of the most common and most deadly scourges of humanity. There is no other which stays so many of our people in the very prime of their career, for it causes one third of the total mortality during the chief working years of life. It kills 53,000 individuals annually in the small population of England and Wales. It is the greatest cause of disablement in adult life. It leads to more loss to the family and to the nation than any other single disease. It is one of the saddest afflic-

tions." Therefore, it behooves all of us, as laryngologists, to be on the alert for the early manifestations if we are to cure tuberculosis in time.

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DISCUSSION.

DR. THOMAS E. CARMODY, Denver, Colo.: After a paper of this kind, it seems almost superfluous to have a discussion, because Dr. Spencer has covered the ground so thoroughly. In the postgraduate course the latter part of the week, Dr. Mayer will go over it and take up the points not mentioned. There are just a few points that I might bring out. Brown gives a percentage of 25, that is in incipient cases, and 45 percent is given by some others in the advanced cases. In U. S. General Hospital No. 21, we found as high as 95 percent showing some incipient involvement. Some had a simple hyperemia, but nevertheless many of these went on to further trouble—ulceration, infiltration and so on—and we must keep that in mind. If we examined all of these cases of laryngeal tuberculosis, we would probably find a much higher percentage. We should always examine cases carefully, and that is done in the sanitariums today, although Mullin's figures show that this was not done in many instances in private work. The larynx was taken care of only when the patients complained.

The point brought out about fatigue of the voice is very important in calling attention to the point that the larynx should be examined. Hoarseness is also of importance, but we frequently do not find this even in very advanced cases, as Dr. Spencer has told you. The interarytenoid sulcus is involved most frequently.

The calcification spoken of by Mullin is likewise important, but we must keep in mind the age of the patient. We may have calcification even when the patient is not affected with tuberculosis. Necrosis, as brought out by Lockard, may be due simply to the tubercle bacillus. Ely claims that we have to have mixed infection. We must always keep in mind that acute and subacute laryngitis must be considered.

As to primary tuberculosis in the larynx, I do not believe it ever occurs. There is a possibility that it might, but with the 96 percent of pulmonary involvement found postmortem, there is a very great doubt. In all the cases I have seen reported, there has been a question about the examination of the lungs and X-ray examination has not been made, or there has not been a postmortem, and this point has not been proved. Lockard's opinion coincides with this.

It is also important to remember the mouse eaten appearance. The punched out appearance of syphilis is not observed, as a rule. It is very infrequently that we find syphilis and tuberculosis combined in the larynx.

(Showed series of lantern slides illustrating various types of ulcers of the larynx).

DR. EMIL MAYER, New York, N. Y.: Dr. Spencer has omitted what to my mind is one of the most important diagnostic points in the early diagnosis, and that is the yellow deposit, canary yellow, that is to be found often in single spots. I have in mind one case, where I made the diagnosis of tuberculosis because the postnasal mirror showed a small exudate on the back of the uvula, which was not as large as my small finger nail. From that the diagnosis of tuberculosis was made, and this was verified afterward by the X-ray. The patient was sent away early and reported after five or six years, and as a result of that early diagnosis the patient is cured. I would like to say in defense of Dr. Spencer, that

in Colorado they are apt to see tuberculous conditions later than we who are clinicians and who see all sorts of conditions in our practice, and so are apt to see these cases earlier.

The question arises as to what we are to do about lupus. Lupus of the pharynx and larynx is undoubtedly a form of tuberculosis. I have seen patients with much destruction of the uvula and larynx, but they were entirely unaware of it, and no bacillus was found until repeated examinations had been made, and then only a single one was found here and there. I shall encroach on my talk for Friday a little, for fear I will not have the necessary time then. In lupus there may be very great destruction. If you will accept lupus as a tubercular condition, with the understanding that when you make a diagnosis of lupus you do not have to send the patient away, it will be an important step. Under my own observation, these patients have lived for eighteen or twenty years. Many years ago, I saw a young girl with tremendous destruction of the pharynx, and made a diagnosis of lupus. She never went away, but married and gave birth to an apparently healthy child. Twenty years later, she succumbed to tuberculosis.

Another thing, in the diagnosis of tuberculosis we are invaluablely helped by the X-ray. That is our great sheet anchor, and I am not prepared to say with our Chairman and many others that primary tuberculosis of the larynx does not exist. If they will accept lupus as a form of this disease, it certainly does. I will show later the thickening between the arytenoids, as also the yellow exudate on the cheek, the tongue and the lip. Sir St. Clair Thomson's name was mentioned as giving a very positive opinion on tuberculosis. It may interest you who have seen him and know what a fine specimen of English manhood he is, to know that he himself had laryngeal tuberculosis. He went into one of the sanatoria of England and spent a year and a half of his life without talking. That rest helped him materially.

We are much indebted to the reader of the paper for bringing up this subject, which is of such tremendous importance and interest.

DR. J. B. McMURRAY, Washington, Pa.: I was very much interested in Dr. Spencer's paper. The Doctor mentioned the symptom of voice tire occurring early in tuberculosis. There must be some reason for this voice tire before we get the intralaryngeal manifestations. I believe there is a symptom that we sometimes see that is of some importance, and that is the condition which we term submucosal edema. You will often observe, early in the day, places where the mucous membrane seems to be lifted a little, with quite deep grooves. This will disappear later in the day. We had an opportunity to observe many of these cases during the war, and it confirmed us in our belief that this is an early sign of tuberculosis. It may be a lymph stasis—I do not know.

DR. FRANK R. SPENCER, Boulder, Colorado, (closing): When I sent my paper to Dr. Levy to look over, he offered some suggestions which he called "constructive criticism," and which were very helpful. He stated that anemia or hyperemia of the larynx is often present before infiltration. He also thought I should put in necrosis of the cartilage as a type before I put in edema.

Dr. Mayer is right in calling attention to the yellow spots. I did not mention findings in the pharynx, but one does find the mottling early.

I think lupus is quite rare in Colorado. I have seen many cases in Europe, and think he has an opportunity in the large clinics in New York to see many. I purposely avoided touching on lupus, and believe the Chairman referred entirely to tuberculosis, when he stated that primary tuberculosis of the larynx is rare.

Dr. Carmody is right about the punched out ulcers of syphilis being rarely seen. I have occasionally seen a punched out appearance that has not been due to tuberculosis.

REPORT OF A GROUP OF CASES OF OTITIC SINUS THROMBOSIS.

SEYMOUR OPPENHEIMER, M.D.

NEW YORK CITY.

Before proceeding to the report of a series of cases of otitic sinus thrombosis seen mostly within the past year in the East, mostly during the prevalent influenza epidemic, it occurred to me that a general resumé of the symptomatology might be in order, as introductory to the clinical histories.

Symptoms. The symptoms of lateral sinus thrombosis are fairly constant, although they may be much masked by the presence of some underlying acute infectious diseases. Temperature is the most important general symptom of sinus thrombosis, and young children particularly are likely to have rises which are excessively high. A perisinus abscess may cause a temperature of a very septic type, i. e., sudden high rises, alternating with sharp remissions; the whole clinical picture may be such as to suggest a sinus infection, but these symptoms may entirely subside after the operative removal of the diseased focus external to the sinus, i. e., a mastoiditis. The mere presence of a high temperature in children is a less important indication for operation than in adults, but when its presence is persistent in conjunction with an acute mastoiditis, and a pyelitis, a pneumonia or an influenza can be excluded, operation is justified even although a blood examination fails to show a bacteriemia. Chills are absent as a rule, although the hands and feet are frequently found to be cold. An important point in a case under suspicion is the taking of two hour temperatures, in order to accurately note the varying oscillations. Typical cases occasionally present themselves, where the temperature remains steadily high without much remission. Headache, pains in the occipital region, and tenderness upon palpation along the jugular vein, due to a lymph node enlargement, may be present. A diagnostic sign of importance, I have observed to be a unilateral enlargement of the lymph nodes at the junction of the facial vein with the internal jugular. The cord like feel along the anterior border of the sternomastoid is a symptom that I have seldom noted, even in cases where the jugular was markedly thrombosed.

Nasal hemorrhage is frequent, due to the fact that the venous blood from the nasal passages is discharged into the superior

longitudinal sinus, and the stasis of the venous circulation in the nose caused by obstruction of the sinus leads to hemorrhage. Drowsiness is the general accompaniment of an acute infectious process, but the state of well being (euphoria) so frequently seen, is oftentimes very deceptive and is apt to throw one off his guard.

Severe persistent headache may excite the suspicion of a complicating meningitis, particularly when associated with vomiting, somnolence and possibly coma. Metastasis is not uncommon. Local symptoms depend largely upon the extent of the sinus affected. Facial cyanosis and dilatation of the temporal and facial veins with epistaxis, suggest an involvement of the superior longitudinal sinus.

Dilatation of the cervical veins and the possible hardening of the internal jugular vein, with or without edema behind the mastoid process, suggest invasion of the lateral sinus. A sign of much diagnostic significance is the presence of postmastoidal edema, indicating a blocking of the mastoid emissary vein.

A symptom, which I believe I have been the first to describe as occasionally present, has been dysphagia.

Upon examination of the throat, there is found a unilateral enlargement of the lymphoid tissue along the posterior fold of the pharynx. This symptom, in connection with the temperature, has in its early stages suggested a possible beginning throat infection as the factor present, but subsequent observation has cleared up this point. In the latter stages of the disease, edema about the orbit and a protrusion of the eye on the affected side indicate an involvement of the cavernous sinus.

It must be borne in mind, that these local signs cannot be relied upon alone as a means of diagnosis, but their close association with a more definite general symptomatology places upon them their dependable value.

The cases presenting the greatest difficulty of diagnosis are those where some acute infectious disease is associated with the otitic suppuration. The temperature curve of malaria is much akin to that of a sinus thrombosis, but in the former case a leucopenia is present, and the blood examination may disclose the malarial parasite.

The external jugular vein on the diseased side may be less distended than on the opposite side, since, owing to the thrombus present in the lateral sinus, the internal jugular vein is less full than on the normal side, and the blood from the external jugular

vein can flow more easily into it. Optic neuritis is present in a fairly large proportion of cases.

Cases of primary jugular bulb thrombosis, when occurring in infants and young children, present typical symptoms, inasmuch as no disease of the mastoid is present. The chief symptom of thrombosis of the jugular bulb is a sudden and rapid rise of temperature in a case of middle ear suppuration to above 104° F., followed by an equally precipitous decline. Thereafter, the temperature curve fluctuates after the manner of the first rise, during which time the variations in the pulse rate follow the temperature. There is usually no distinct chill, but the hands and feet may be cold when the temperature rises. Later on, when the bacteriemia becomes more pronounced, prostration ensues and all the symptoms of sepsis become apparent.

Fundus examination may show a neuroretinitis in some cases. Crowe, of Baltimore, lays some stress from the diagnostic standpoint, on being able to produce choked disc by compression of the internal jugular vein. I have never been able to corroborate this test.

In every case of mastoiditis, lateral sinus thrombosis is always a possible complication. Its relatively high mortality, with the absolute necessity of prompt operative intervention to prevent a general pyemia, demands that all factors that will in any way aid in its early recognition should receive the most careful attention.

In establishing the diagnosis of sinus thrombosis, it is of course essential that all other diseases which might cause a like symptomatology should be definitely excluded. Among such may be mentioned pneumonia, typhoid fever, acute endocarditis, malaria, and certain cases of scarlatinal infection. Their distinguishing features frequently demand the close cooperation of the otologist and the experienced clinician.

In infantile hemiplegia, sinus thrombosis should be considered. In a series of 78 autopsies, reported by Starr and Westcott in infantile hemiplegia, sinus thrombosis was found in 5. All cases require an exhaustive consideration of the entire symptomatology, and above all the taking advantage of the aid given by blood cultural examinations.

A high temperature continuing several days after a mastoid operation, especially when the operative findings have disclosed areas of necrosis of the bony covering of the lateral sinus, and an examination of the blood showing a bacteriemia, is indicative of an infective process constituting a sinus thrombosis, and de-

mands prompt exploration of the sigmoid sinus. An occluding thrombosis occupying the lateral or sigmoid sinus may exist without producing any symptoms referable to the internal jugular vein.

Blood examination furnishes reliable data in many cases. In the early stages of thrombosis, the blood shows as a rule an increase in the number of white cells. The increase is rarely over 20,000. When the thrombus is infected or suppurating, a bacteriemia may be demonstrated by blood culture, but in many cases a clinical diagnosis may be made before a positive blood culture can be obtained. Frequent reports upon the blood examinations in these cases have proven that thrombosis may exist without a characteristic blood picture, but a positive blood culture is, of course, absolute evidence that the pathogenic organisms have entered the circulation, and constitutes an indication for immediate operation. A negative blood culture, however, does not necessarily mean that the sinus is not involved.

In studying the bacteriologic flora in a large series of cases of aural infection which came to operation, the streptococcus or streptococcus mucosus was found in all.

In smear examinations of aural discharges, streptococcus is probably the most frequent of various organisms. Its differentiation from the pneumococcus is at times difficult, particularly in the light of Rosenow's contention as to their transmutation.

In a previous communication, the conclusions reached, were that the detection of a bacteriemia should be possible in every case of sinus thrombosis at some time during the course of the disease, although it is possible, that as the result of various causes, such as a sterile thrombus situated below the infected clot, the bacteriemia might not become evident for a time at least, or the culture may be taken before the bacteria are thrown off into the circulation, whereas if it were taken a few hours later the organisms would be found.

After a mastoid operation, where sinus thrombosis is suspected, but where symptoms are not sufficiently definite to warrant opening the sinus, and a blood culture has given negative results, it is most essential that subsequent cultures be taken. The rule can be laid down, that in the presence of streptococci in the blood stream, there is a septic focus and further operative measures are necessary.

In a case where the sigmoid sinus has been attacked, but the jugular vein has not been ligated, the persistence of a positive blood culture is an imperative indication for ligation of the jugu-

lar vein. Should positive cultures remain after the ligation of the jugular, it would be suggestive of a bacterial infection of the endocardium or a metastatic process in the lung.

In an experience dealing with approximately 160 cases of sinus thrombosis, I have never observed an infection due to any organism other than the streptococcus or the streptococcus mucosus. This point has proven in many instances of great value in the expression of an opinion, and shows the importance of a culture from the pus contained in the mastoid process at the time of operation.

In a number of these cases, the mastoid infection was of the pneumococcus type. After the mastoid operation, symptoms presented which were suggestive of a complicating sinus thrombosis. Blood cultures were negative. Operation was not advised on the sinus in these cases, and a pneumonia, an erysipelas or some other complicating condition developed, which explained the symptomatology so much akin to that of a sinus thrombosis. One can see, therefore, the diagnostic significance of a negative blood culture in connection with a nonstreptococcus type of infection.

REPORT OF CASES.

F. D., male, aged 34 years. On March 20th, right otitis media suppurativa with myringotomy. Aural discharge very scant for ten days. At work with no apparent symptoms until April 20th; then chills, high temperature, and swelling of left knee. Under the care of his physician, with no improvement. April 30th: Case seen in consultation with a general surgeon, who expressed the opinion that the arthritis was not primary, but secondary to some other focus. Admitted to Hospital; temperature 102° F., white corpuscles 13,300, polynuclear count eighty-two percent. Patient looked septic. No aural discharge and membrana tympani slightly reddened and thickened, but like a resolving process. No mastoid tenderness, no intratympanic evidences of mastoiditis, and slight submaxillary and cervical lymph node enlargement. Left knee joint showed existence of an acute synovitis and peri-arthritis. Blood culture, in 24 hours, positive with 45 colonies of streptococci. Aspiration of left knee joint; large amount of yellowish purulent material containing streptococci. May 1st: Mastoid operation. Cellular structure macroscopically normal, with no evidence of disease except on reaching the sinus groove, when some pus was encountered. Sigmoid sinus was uncapped, the dura found thickened and covered with plastic exudate. At the bend of the sinus knee, the dural covering was ulcerated, and

a protruding partially broken down thrombus was found. The jugular was ligated and exsected well below the thrombus, which extended to the level of the facial vein. The external sigmoid sinus wall was removed, the exposure being carried to within a very short distance of the torcular. Patient's condition most satisfactory. May 2nd: Blood culture still positive. For the next eight days, repeated blood cultures continued positive, but the patient's general condition was most satisfactory and temperature was low. Wounds satisfactory. I might add that the number of colonies of bacteria in each c.c. became steadily less, until on May 10th there were but three on the plates, with none in the flask. May 10th, temperature rose to 103° F., and pain and swelling were observed in left shoulder. From this time until May 15th, when the patient died, temperature ranged from 102° to 104° F., with some meningeal symptoms. Eyegrounds showed retinal changes and papillitis. Lumbar puncture: fluid under great pressure. Culture of cerebrospinal fluid negative, but cells increased. Fehling reduction marked. May 12th: Subdural drainage in the temporal and anterior cerebellar region. During the course of the disease, the patient manifested petechiae of the left lower conjunctiva and small reddish nodules on the soles of both feet. Heel of left foot painful, swelling and tenderness of right knee, right and left shoulders, and bullae on fingers of right hand. Temperature before death rose to 107° F.

Postmortem. I will refer only to the examination of the heart, which showed on the mitral valve, localized on both flaps, on the auricular surface near the edge of insertion, two areas of cauliflower like grayish vegetations, each 1 cm. in width, one of them projecting several mm. and the other with a knob like protuberance about one cm. in width and diameter. The remainder of the heart was normal. Smears from vegetations showed streptococci. This finding explained the continuation of the positive blood cultures after the jugular resection. Repeated physical examinations by the medical attendants had failed in the recognition of this condition, which by reason of the foregoing they had suspected.

C. R., female, aged 49 years. Aural discharge for fifteen years. Three months ago, discharge ceased followed by postauricular swelling, which with local applications of ice speedily became reduced. One month later, swelling of ankle joint, for which she consulted a physician, who found in addition a pericarditis and endocarditis with pleural exudate. After three weeks, swelling from ankle having subsided, patient went to work,

which was followed in a short time by recurrent swellings of ankle joint and considerable dyspnea, which confined her to her bed for two months. Sudden pain in the ear was then complained of, with a chill and rise of temperature to 104° F. The following day another chill and rise of temperature to 104° F. At this time I first saw the patient. Local examination: Membrana tympani absent, with some granulations in the middle ear, and slight odorous discharge. Mastoid not sensitive. No choked disc. Diagnosis, sinus thrombosis. Operation findings: Extensive destruction of inner table of mastoid, particularly of posterior fossa wall. Large perisinus abscess with erosion of sinus wall. Sinus contained a purulent disintegrating thrombus. Dura over cerebellum covered with dirty granulations. Jugular ligated and excised. From below the level of the facial vein, the jugular was completely obliterated and was recognized only as a cord. Along its sheath were many enlarged glands. For two weeks, patient did well, and then symptoms of cerebellar abscess became manifest, and operation discovered a large abscess of occipital lobe. Death followed with evidences of a leptomeningitis. Autopsy refused.

J. J., female, aged 14 years, admitted to medical service of Hospital with diagnosis of acute articular rheumatism. Two years previously, an attack of acute articular rheumatism came on shortly after an acute suppurative otitis. Two months before admission, acute aural suppuration, lasting for four weeks, then completely subsiding. For one month, chills almost daily, high temperature, sweating, vomiting and headache. Swelling of phalanges of left hand and left hip joint, and stiffness of neck. Blood culture positive. Patient transferred to otologic service. No aural discharge. No mastoid tenderness. Beginning optic neuritis. Operative findings: A purulent thrombus in the sigmoid sinus and upper part of the jugular vein.

A. L., aged seventeen years, under my care for apparently a very mild acute suppurative otitis, postinfluenzal. Myringotomy; culture. Streptococcus mucosus. On the tenth day, ear symptoms ceased and I discontinued attendance. A few days later, pain and swelling in phalanges and wrist of left hand, followed within the next week by a rapid development of involvements of both knees, both elbows, right hip, and right shoulder joints. Aspiration of knee and shoulder joints; no bacteria recovered. Severe distressing bronchitis, not relieved much by medication, nor did the inflamed joints respond to treatment. This condition continued for six weeks with no apparent ear symptoms, but

with moderate temperature. Then mastoid tenderness, a sharp chill, and temperature of 103° F. Ear examination: Membrana tympani intact, but thickened and reddened. Tenderness and edema of mastoid, and sagging of membranous canal wall. Diagnosis: Mastoiditis with sinus thrombosis. Operation: Beginning perforation of mastoid cortex and internal destruction, particularly along sinus plate. Pus from mastoid showed streptococcus mucosus. Thrombus from sinus, partly disintegrated in central portion, extended into bulb and below, to about level of the thyroid vein. Jugular ligation and exsection. Recovery.

R. S., male, aged forty-one years, under care of physician for two months with swellings of ankle, knee and both shoulder joints, with no improvement from a large variety of medication, temperature varying from 99° to 102° F. Complained of pain in ear, for which I was called in consultation. Local findings: Slight swelling of auditory canal, mastoid slightly sensitive. Foul smelling discharge, scant in amount. Ossicles necrotic and membrane deficient. History elicited of chronic aural suppuration with few external symptoms, described by patient as being but a slight moisture. Temperature 105° F., preceded by chilly sensation. Diagnosis established, after one day of observation, as sinus thrombosis. Mastoid operation: Sclerosed mastoid with few granulations, but no free pus. Sinus plate necrotic over a very small area. Dura of sinus covered by thickened exudate and granulations. Sinus contained a firm, white, apparently well organized thrombus, streptococci being demonstrated in thrombus and vein wall. Jugular ligation and exsection. Recovery.

The preceding five histories demonstrate that an otitic sinus thrombosis may have its clinical history completely masked by the existence of an acute arthritis. In the second case, the joint symptoms were associated with an endocarditis which improved sufficiently to allow the patient to go to work, and was followed by a relapse of the joint swelling. In two of the other cases, a number of joints became involved in turn, which, strange to say, were in a measure improved by specific remedies, both in the retrogression of the temperatures and the local swellings. In the fifth case, the attending physician who summoned me at the time of the ear pain, pronounced the case to be one of obstinate acute articular rheumatism. In this case, there had been an old suppurative otitis, but the arthritic symptoms had been manifest for two months before evidences of a sinus thrombosis. In the

second case, three months passed before striking evidences of a sinus thrombosis appeared, the clinical picture having been entirely dominated by the joint swellings and the inflammation of serous surfaces. It was only my first experience which caused me in the other cases to refer the arthritic swellings, appearing after a middle ear infection, to a sinus thrombosis.

In the fourth case, aspiration of the joint failed to find bacteria. This experience in other cases leads to the conclusion that it is unjustifiable to presuppose that pyogenic cocci are present in all secondary joint manifestations, otherwise we should observe abscesses of the joints much more frequently in bacteriemic processes such as those under consideration.

From our present knowledge, I believe that toxins can be designated as the cause of such bacteria free exudations.

On perusal of the literature, I find only two cases reported which bring articular rheumatism into etiologic relation with otitic disease; the report is by Wolff, who studied two cases of acute middle ear infection following a typical articular rheumatism. In one, a cardiac lesion developed from which the patient died one year later. In both cases, numerous joints became involved, as in genuine acute articular rheumatism, and in the second case a relapse occurred after six months, but cleared up. It is noteworthy that the otitic symptoms were very mild, and the mastoid showed no evidences of involvement. Wolff is of the opinion that both his cases were of genuine acute articular rheumatism, and that the specific excitors of the disease had settled in the tympanic cavity, similar to lodgment in the tonsillar structure, and therefore the acute otitis must be considered as a symptom of acute rheumatism, and not as a cause. As a matter of fact, however, a close analysis of his cases shows that from the first the acute ear symptoms appeared, followed in a few days by the symptoms of joint inflammation with a sharp rising temperature, rather too high for an uncomplicated middle ear infection. It is possible that in his two cases a sinus thrombosis developed, which healed spontaneously by obliteration. One of my cases corresponds to his in the fact, that after myringotomy only the scantiest secretion took place, and later, at the time of the mastoid operation, a perforation of the membrana tympani, if present, was so insignificant that it could not be recognized.

Our contention resolves itself into the following: By no means is it contended as unlikely, that after an acute middle ear infection symptoms of acute joint inflammation may not arise, which would then have to be considered as a symptom, but if,

after an acute otitis or during the course of a chronic suppurative otitis, acute arthritic symptoms are present, they must be considered not as a primary, but as a secondary metastatic complication, resulting from an infective otitic phlebitis and an associated general bacteriemia.

It is interesting to learn that an ear disease is able to call forth the clinical picture of a genuine acute articular rheumatism, and that the ear symptoms may be so slight, and by contrast with the joint condition be pushed so far in the background, that the otologist runs the risk of overlooking the underlying cause, thereby exposing the patient to the danger of an unfavorable outcome, for in the great majority of cases, an unoperated sinus thrombosis runs a fatal course. In view of these facts, it does not seem amiss to suggest to bear in mind in all cases of arthritic inflammation with an associated otitic lesion, that the latter is likely a primary causative factor.

E. B., age 15 months. Gave a history of three weeks' acute ear infection, postinfluenzal, with a number of myringotomies. Condition on examination showed an involvement of both mastoids. Operation was immediately performed. Temperature 105°. The child has a curious high pitched meningeal cry. Nothing unusual in the findings of the operation. The sinus was exposed on the left side. The temperature varied for the ensuing seven days from 101° to 103°, never reaching normal. During this time, the symptoms of photophobia and facial twitching were noted. At 3 a. m. on the morning of the 7th day, a tremendous hemorrhage took place, from the left side. The child was promptly seen and found to be almost completely exsanguinated. 400 cc. of the mother's blood was transfused, with marked improvement. The following day the temperature rose to 105°. It was reasoned that there was no incompatibility in the blood of the donor and recipient, and that the rise of temperature was due to the development of an infective phlebitis. It was argued that if a hemorrhage from the sinus had taken place as the result of an erosion of its wall, an infection must be present in the lumen of the vessel. Operation was therefore performed, consisting of a primary ligation of the jugular vein without a preliminary exposure of the sigmoid sinus. A well organized thrombus, extending well down into the jugular bulb, was removed, as well as a large thrombus which was washed out from the torcular end. The temperature rose after the operation to 106°, but gradually dropped, with uneventful convalescence.

Comment. The interesting question that arises is just how we should handle a mastoid wound, where there has been an erosion of the bony plate over the sinus. If we employ tight packing, it being a field that cannot be made surgically sterile, it is quite possible by pressure to induce a slowing of the blood stream through the sinus, and also to cause a trauma of the dural wall, with the induction of an infected phlebitis. If, on the other hand, we pack the cavity too lightly and there is an infection of the sinus wall, this may rupture from ulceration, as it did in this case, and the packing would be insufficient to hold this tremendous hemorrhage in check.

A. A., female, aged twelve, was seen by me in consultation, with a history that for two weeks previously she had had a slight cold, but felt well enough to be allowed to go to school. The mother noticed a slight discharge from the ear during this period. Two days prior to my examination, the child complained of pain in the left ear, which became more pronounced during the day. The following day, the temperature varied between 102° and 103°; pain in the ear less, with some discharge. Patient in bed and comfortable. During the night, the child became irritable and complained of much pain. The day of my examination, the child was very irritable and restless and complained of headache; discharge from the ear profuse; temperature 104°.

At 10 a. m., the child had a pronounced chill and gradually sank into coma; at 11 a. m. she was completely unconscious. At 1 p. m. I first saw the child. Temperature 106.5°. Patient comatose, the local condition presenting all the evidences of a marked, acute mastoiditis, and the physical state indicating an intracranial involvement. No convulsions had been noticed; slight generalized spasticity of the extremities: double Kernig and McEwen's signs present.

The patient was removed to the hospital and immediately operated upon. The usual curvilinear incision was made through the skin and periosteum over the mastoid process; the entire mastoid process was involved in an acute infection of a very severe type; the bone was of a greenish-yellow hue, cells infiltrated with a thick greenish fluid. Culture showed streptococci. The entire mastoid process was rapidly removed, the extensively necrosed tip being removed en masse; the sigmoid sinus plate was found necrotic, its removal exposing the sinus wall. The vessel was contracted and firm, and appeared thrombosed. The mastoid emissary vein was exposed and traced to its ending in the sinus; it also was thrombosed, standing out in sharp relief with its thickened walls.

On removing the cells over the mastoid antrum, a fistulous tract was seen through its roof, through which pus escaped. The opening was enlarged and about two drachms of greenish pus under great tension was liberated. The dura was then further exposed, by extending the skin incision anteriorly over the ear and removing a large plate of bone over the squama of the temporal bone, including the antral and tympanic tegmen. The dura was then seen to be markedly congested and bluish-black, bulging pronouncedly. The dura was freely incised; the presenting brain was much congested and showed over a large area a mushy friable appearance, with exudation. The piaarachnoid was greatly congested. Fresh adhesions between piaarachnoid and dura were bluntly separated, and a gauze drain inserted into the subdural space. The brain was pulsating, bulging and markedly softened. The vertical limb of the sigmoid sinus was then compressed above and below, in the site of its exposure, and incised. A fresh reddish-brown clot presented immediately, after which there was an escape of blood. Free hemorrhage was established from the distal and proximal ends of the vessel. Blood culture showed four colonies of streptococci to each cc.; polymorphonuclear count 90%; white blood count 31,000.

March 4th, day after operation: Patient in a state of wild delirium; temperature 104°. Lumbar puncture practised for the relief of intracranial pressure; cerebrospinal fluid under great tension and cloudy, but bacteriologically negative. Polynuclear 75%, mononuclear 25%. Albumin increased. Prior to the receipt of the laboratory report of the cerebrospinal fluid, anti-streptococci serum was injected into the spinal canal.

March 7th: Temperature 106°; patient still delirious, cannot be roused except with difficulty; lumbar puncture; fluid clear, but still under great pressure. Blood culture now sterile.

March 8th: Temperature 103°. For the first time, patient is somewhat rational. Blotchy erythema present, but quickly disappearing, leaving large areas of localized edema in its wake.

March 9th: Temperature 101°. Decrease of leucocytosis and polymorphonuclear count. Mental condition better. Large protrusion of necrotic brain substance through dura; some indefinite symptoms of aphasia present; exact character impossible to determine, owing to the general restlessness of the child. No attempt was made to cut away any of the necrotic brain tissue, as it was feared further injury might be done to some of the brain centers. Subdural drainage discontinued. Large quantity of cerebrospinal fluid has been steadily escaping.

March 12th: Temperature near normal. Dressing of wound followed by a giant urticarial rash, quickly disappearing; also edema of eyelids, lips and vulva, which persisted for some hours. General condition good, but child extremely irritable.

March 15th to 18th: General condition steadily improving. Definite evidences of optic aphasia present, which however appeared to be transitory; eyegrounds negative.

Examination of aphasia, four objects being used, a pencil, penny, knife, watch. Recognizes objects seen and can indicate their use by appropriate motion. Does not recognize objects felt. Recognizes taste and smell. Cannot name objects seen. Cannot name objects felt. Understands speech perfectly. Does not understand printed and written words. Cannot read aloud printed or written words and does not seem to understand the printed or written words. Can write the name of objects seen, but only when their names are told to her (i.e., can only write at dictation). Can copy perfectly. Power to speak voluntarily is fair, words being occasionally jumbled or transposed. Can state all her wants very well. Repeats words perfectly.

Night of March 18th: Patient became extremely irritable and rapidly sank into a stuporous state. Temperature rose sharply to 104° . Polynuclear count 84%. Lumbar puncture immediately practiced; large quantity of turbid fluid withdrawn under great pressure. Smears and culture of fluid negative. Polymorphonuclear 5%; mononuclear 95%.

March 19th: Temperature 105° ; condition unchanged; patient very noisy and unmanageable.

March 20th and 21st: Temperature down to 101° ; general condition markedly improved, although another rise to 104° followed on the 23rd.

The condition of the past five days was attributed to the local encephalitis rather than to a general cerebral process, which contention is justified by the pronouncedly apparent change for the better in the child's general mental and physical state, which subsequently took place.

April 1st: Condition has gradually improved; temperature normal. Much difficulty was encountered in managing the hernia cerebri, which, however, by systematic and gradually increased pressure was forced back into the cranial cavity. Aphasia gradually improving.

April 20th: Aphasia now limited only to a loss of ability to remember names of certain objects, and loss of the retentiveness of memory.

Improvement from this time on was uninterrupted, and while it was thoroughly realized throughout the course of the illness that a developing encephalitis might at any moment defeat all our surgical efforts, yet much gratification was felt at the outcome of a case, which can only be described as moribund at the time of the primary operation.

B. K., age 18. Discharge from left ear for three weeks following an acute head infection. All of the evidences of an acute mastoiditis were present, and the patient was promptly subjected to surgical interference. The presence of a slight facial paralysis, as well as a slight paralysis of the external rectus muscle, were symptoms present which are not in line with an uncomplicated mastoiditis, but the operative findings did not throw any light explaining their presence. Eleven days after operation, the patient left the hospital with the wound granulating nicely. The subsequent dressings were then carried out by an associate, who advised me two weeks later, that the patient had been running some temperature, ranging as high as 104° for a few days, and complained of severe frontal headache. A suggestion of a chill had been present. I had the patient returned to the hospital for observation, having of course in mind the possibility of a complicating sinus thrombosis, but as such a complication would be rather unusual four weeks after the primary mastoid operation, there was as well considered the question of a possible influenza, the pandemic then being so prevalent. The facial and external rectus paresis was still present. For the ensuing five days, the patient was kept under close observation; the temperature range was not sufficiently definite to suggest a sinus thrombosis, in fact it seemed to show a receding tendency. Blood culture was negative as well as a Widal, and the blood count was within normal limits. Eyegrounds normal. The state of euphoria was most striking. A second blood culture, taken three days after the first, with the general physical state of the patient improving, was reported as positive. An operation was then undertaken upon the sigmoid sinus. There had been no exposure of the sinus at the time of the primary operation. The dura over the sinus appeared normal until the exposure reached the knee, where it began to show some thickening. Under the usual technic, with compression plugs above and below, the exposed sinus were incised. The lumen contained a well organized thrombus, which extended to within a very short distance of the torcular. The overlying bone was removed in order to remove the thick external sinus wall, and in order to establish free bleeding from its end. The bulbar end

of the sinus was then attacked, but as bleeding did not take place after very mild curetting, the jugular vein was exposed in the neck and ligated close to the clavicle, and above and beneath the angle of the jaw, as well as its tributary veins, all of which were exsected between the ligatures. The neck portion of the vein contained no thrombus. There was some slight lymphoid nodular swelling present along the sheath of the vein, but particularly at the junction of the facial vein with the internal jugular. The neck wound was closed with the exception of its lower end. A blood culture, taken within a comparatively few hours after the operation, was negative. The convalescence was uneventful. The organism recovered from the second blood culture was streptococcus hemolyticus, the same organism being recovered from the thrombus and the exsected wall of the sinus.

Comment. The extremely interesting features of this case was the late development of the sinus thrombosis after the original mastoid operation, and the improving condition of the patient in the presence of a blood vessel infection of unusual degree, as manifested by the extent of the pathologic lesion.

A. S., age 16. Two years previously, I performed an acute mastoid on the left side. During the winter contracted pneumonia, during the course of which acute ear symptoms developed, for which a myringotomy was performed. An acute mastoiditis developed on the right side. A severe pansinusitis was present as well. The appearance of patient rather dusky and cyanotic, probably due to the influenzal pneumonia still present. Temperatures very high. Mastoid operation showed nothing unusual. Culture from mastoid streptococcus hemolyticus with marked involvement of all cells. The temperature rose after the operation to 105°, and varied for the next three days from 102° to 104°. Wound dressed four days after operation, and it looked unusually dry, some edema being present about the posterior part of the mastoid and temporal portion of scalp. No neck symptoms, no headaches, no chills, but euphoria marked. Patient makes bad impression; the eyegrounds show slight engorgment of retinal vessels; no meningeal symptoms. Diagnosis tentatively made of sinus thrombosis. An exploratory operation performed preceded by a transfusion of 600 cc. of blood.

Transfusion. An attempt was made to do the agglutination and hemolysis tests, but it was impossible to collect blood in citrat by means of a puncture wound of the finger. The red blood cells immediately formed large microscopic clumps. Under the microscope, the red cells of the patient's citrated blood ap-

peared agglutinated. It was, therefore, impossible to judge the agglutinative powers of the donor's serum on these cells. However, since the professional donor with us was a group four donor, the transfusion was begun omitting the tests. 500 cc. were transfused preliminary to the operation. A blood culture was taken before operation.

The sinus was exposed for the entire distance of its sigmoid portion; dura covering sinus appeared absolutely normal and the sinus patulous. In the removal of the bony wall, the sinus bled tremendously freely, from which it was assumed, in view of the normal appearance of the sinus and temperature not being characteristic of a sinus thrombosis, that a thrombus was not present, or if present was of a parietal nature, and was washed out and not discovered in the enormous gush of blood. I decided, in view of the above, not to ligate the jugular unless the blood culture was reported as positive. Lumbar puncture negative. After the operation, the blood culture was reported negative. For two days following, the patient apparently better, but the temperature continued to range 102° to 104°. The following day the patient appeared much more sick. Blood count showed a poly count of 95%, and a white count of 21,000. Another blood culture was taken, and reported a few days later as negative on aerobic plates, but as positive on anaerobic plates. We were evidently dealing with a typical type of streptococcus infection, and the case shows the importance of taking cultures on both types of plates. The temperature rose to 106°.

Operation followed by transfusion. The same condition of the blood collected in citrat from a puncture wound of the finger existed as previously. Here again the transfusion was attempted, in spite of the fact that the tests were omitted, inasmuch as the donor was a group four donor. 800 cc. were transfused after the operation had been performed. The jugular ligation was performed with great difficulty, as the vein in the neck was much matted to the neck tissues, due to the inflammatory process about the sheath of the vein, with much lymph node enlargement, making its locating very tedious. Transfusion was followed by hematuria either due to an incompatibility of the donor or to toxic origin. A blood culture taken at this time, aerobically and anaerobically plated, showed, after 12 hours' incubation, on the anaerobic plates a growth of about forty green producing colonies, with no evident hemolysis around them. The lower portions of the broth cultures were cloudy, whereas the upper strata of these were clear. Upon staining the colonies from the anaerobic plate,

a long chain streptococcus was found in pure culture. Twelve hours later, the aerobic plates showed about fifty colonies, each of the same variety as was found in the anaerobic plate. At the operation, the sigmoid portion of the sinus was incised, and no thrombus was present, but a wire ring curette introduced into bulb removed a small well organized parietal thrombus. The patient was much cyanosed after operation, and the temperature dropped to 101°, rising the following day to 106°. Death then ensued. A blood culture, about twenty hours after the jugular ligation, remained markedly positive, suggesting the possibility of a bacterial endocarditis being present.

Comment. The interesting points of this case are particularly the undoubted clinical evidence of a sinus thrombosis, which was not determined at the time of the operation upon the sigmoid portion of the sinus, its normal physical appearance, and the curious cultural characteristics of the organisms, their growing on the anaerobic plate but not on the aerobic, which I think demonstrates the importance of possibly having cultures taken on two sets of media.

J. D. W., female, aged six years, was seen by me in consultation on the sixth day of a scarlet fever, on account of a profuse mucopurulent discharge from the nose and a severe throat inflammation. There was marked submaxillary and cervical adenitis, both nasal and faucial conditions presenting all the evidences of diphtheria, but culture failed to show the Klebs-Loeffler bacillus, streptococci being reported. The ears were also examined at this time, a slight congestion of the right tympanic membrane being present; temperature 103°.

Three days later, spontaneous perforation of the drum membrane took place, without any definite reference being made to the ear by the child. At this time, I again saw the patient and found a large perforation of the drum membrane, with profuse sero-sanguinous discharge; no mastoid tenderness; nasal and faucial condition as well as the adenitis markedly improved. The culture of the aural discharge showed streptococci in abundance and a few staphylococci. The temperature varied between 101° and 104°, at which it remained for the ensuing six days. During this period, the local otitic condition became pronouncedly worse, with all the evidences of a developing acute mastoiditis.

On the night of the 25th of February, on the 11th day of the otitis and the 17th day of the scarlet fever, the mastoid process was opened. The operative findings showed unusually well developed interosseous cellular structures for a child of this

age. The mastoid cortex was very thick. Considerable free pus was present in the mastoid antrum, the tip of the mastoid process being markedly necrotic. A macroscopically normal plate of bone covered the sigmoid sinus, which was not removed, no exposure of the underlying dura occurring. The temperature following the operation dropped to 99° and immediately rose to 103.8°. This was considered at the time a not unusual postoperative rise. After another slight remission, the temperature steadily rose for the subsequent 48 hours to 105°. The little patient was extremely fretful and peevish, and complained of occasional chilly sensations along the limbs and back. In the next 24 hours, two additional rises of temperature to 105° took place, with sharp remissions to 100°, making altogether three rises to 105° within one day. Thorough exploration of the wound showed no local infective process. Small pink granulations were springing up, less marked over the bony sinus plate. The wound looked rather unusually dry, the dressings not being soaked as profusely as customary. During the period following the mastoid operation, there had been a progressive increase in the polynuclear count from 72% to 84%, leucocytosis also increasing. Blood culture negative. All other general conditions having been excluded, the diagnosis of an infection of the sigmoid sinus was made, being based chiefly upon the sharp, rapid temperature oscillations, preceded by what is equivalent, in a child at least to a chill, i.e., complaining of being cold. The maintenance of the persistently high temperature after the mastoid operation led me to believe that an infective phlebitic process probably antedated the before mentioned procedure.

Under chloroform anesthesia, the bony plate over the vertical limb of the sigmoid sinus was removed, making a large exposure from a point beyond the sinus knee to a point as close to the jugular vein as possible. The exposed sinus wall was decidedly thicker than normal and was wrinkled in folds, nor did it protrude from its lateral bony attachments as pronouncedly as does the normal vessel when the overlying bony plate is removed. But little attempt was made at palpation, as I am of the opinion that the likelihood of dislodging of a parietal thrombus into the circulation is quite possible, unless definite occlusion can be made of the vessel at its proximal end. Incision of the sinus wall was followed by a very undetermined flow at first, which in a few seconds was followed by profuse bleeding. The thrombus was not found, although the slow character of the bleeding following the incision, followed shortly by an increase in the volume of the

stream, leads me to believe unquestionably that a parietal clot was present, which was washed out by the subsequent gush of blood.

Free hemorrhage being established, both from the distal and proximal ends of the sinus, the external vessel wall was incised, and the wounds packed in the usual manner. Immediately following the operation the temperature dropped to 99°, and with slight variations has since remained normal, the general mental and physical condition of the child at once pronouncedly improving.

F. A., age 8. Left ear discharging for 4 years. Two weeks ago, the child's neck became rigid, it could not stand, and complained of pain in knees. Vomited. Has had persistent high temperature, and continuous headache.

Examination. Child pale and in pain. Emaciation marked. Left ear discharge. Drum perforated, thickened. Tenderness left side of neck.

Blood culture. Hemolytic streptococcus.

Operation. Mastoidectomy. Incision of sigmoid sinus. Excision of jugular vein. Usual curvilinear postauricular incision. No cellular structure found in mastoid. Sigmoid sinus exposed and appeared thickened; lower end perforated and bathed in purulent material, and disintegrated thrombus. Incision along anterior border of sternomastoid. Jugular vein adherent throughout entire course to muscular planes and carotid sheath. Vein freed and incised. Thrombus extended well down beneath the exposed lower end of vein, and it was impossible to establish a return flow. Quantities of semiorganized clot withdrawn, and ring curette introduced into lumen of vein extending down into chest. Small gauze drain introduced into vessel. Return flow established from torcular. Free flow from upper end of incision. Probe passed down jugular way down into great vessels, but no return flow established. After operation, temperatures remained very high, reaching 106°. Three injections of Hiss' leucocytes extract given with no effect, also repeated transfusions. Child very restless and noisy, also in much pain, and gradually became comatose on the 2nd day and died. Repeated blood cultures all negative.

Postmortem. Body is that of a poorly nourished female child. There is a marked prominence and distention of the superficial veins over the chest, especially the veins on the left side of the chest. There is a partially healed surgical incision over the left mastoid which is healthy in appearance, and another partially healed incision in the lower portion of the left side of the

neck, just above the clavicle, and over the anterior margin of the sternomastoid muscle. This lower incision was reopened, and the tissue found edematous and fibrous. Beneath the anterior margin of the sternomastoid, about three centimeters above the clavicle, was found a small abscess cavity, one centimeter in diameter, filled with cream colored pus. A probe inserted into this cavity entered the lower segment of the internal jugular vein. The jugular vein, between this situation and just below the mastoid, had been removed at the operation. The left jugular and innominate veins, and a portion of the right innominate vein, the superior vena cava, and the heart were removed in one piece. The left jugular vein was opened and its uppermost portion was found practically completely obliterated by an almost decolorized adherent thrombus. Below this, and almost as far as the junction of the superior vena cava, the veins were lined by adherent, almost decolorized thrombus, but their lumina were only slightly constricted. The thrombus was from two to three millimeters in thickness. There is no thrombus in the right innominate vein, and the heart showed no abnormality.

Sigmoid sinus. The mastoid wound was reopened, and a large portion of the temperoparietal and occipital bones removed. The upper end of the left jugular vein, the sigmoid sinus and the transverse sinus, almost as far back as the torcular, along with the dura surrounding them, was removed in one piece. An area of the sigmoid sinus, about two and one half centimeters in length, was exposed in the original wound. The sinus beneath this was practically completely obliterated by an almost completely decolorized thrombus. In the jugular vein below this, there was also found an almost completely decolorized thrombus, obliterating the lumen. In the transverse sinus, above the sigmoid sinus, as far back as the torcula, a mixed red and gray thrombus was found adherent in places, and almost completely obliterating the lumen. The petrosal sinuses could not be investigated, because of the nature of the autopsy.

Brain. The cerebrospinal fluid was thin and cloudy. A portion of the left cerebrum, which was removed, showed marked edema and cloudiness of the meninges, and a fibrinous deposit in the meninges along the sulci.

Lungs. Were inspected, and no infarcts were found. They were mottled in appearance with small red areas in the pleurae.

Microscopic examination. Thrombus in innominate vein: Chronic inflammation in the wall of the vein. There is an almost completely organized thrombus deposit upon the intima.

This is an almost entirely blood platelet thrombus, and is well along in the process of organization. Gram-Weigert stain shows a moderate number of cocci, occurring singly, in pairs and in chains in the thrombus.

Thrombus in transverse sinus. Is almost entirely a blood platelet thrombus, showing well advanced organization around its peripheral portion. The center is necrotic. Gram-Weigert stain shows very few bacteria, which are probably contaminations at the time of autopsy.

Smear of thrombus from upper portion of left innominate vein shows amorphous material and a moderate number of Gram positive cocci, some of them in short chains.

Smears of thrombus from lower portion of left innominate vein shows a moderate number of Gram positive cocci, but more than in smear from thrombus in upper portion of left innominate vein.

The following case presents several points of great interest, and especially does it seem of importance on account of the destruction present in the interior of the temporal bone, with involvement of the sigmoid sinus, and yet, at times during the course of the disease, the symptoms were so slight as to scarcely attract attention.

A. L., aged twelve months, had a severe coryza when eight months old, accompanied by a marked degree of nasal stenosis, to such an extent that it was necessary before each nursing to cleanse the nasal chambers with an alkalin solution and reduce the congestion with adrenalin. At the end of one week, the coryza had almost disappeared, and he was in good condition, when the temperature rose to 102° F. after a restless night. A thorough examination revealed nothing abnormal except the left ear, which showed marked congestion of the membrana tympani and bulging of its lower segment. A free incision was then made in the tympanic membrane, and an excessive amount of pus was then released under pressure. Within a few hours the temperature had returned to normal, and the child showed no evidence of pain or discomfort in any way. On account of the profuse discharge, the canal was carefully cleansed twice daily, and gauze drains were employed, so that at the end of ten days the discharge had entirely ceased, the incision in the tympanic membrane had healed, and in all respects the child appeared to be perfectly normal.

Nothing was then seen of the patient for nearly four months, when the following history was obtained. He had remained per-

fectly well until five weeks previously, when following a slight coryza, he became very restless, seemed to have considerable pain in the previously affected ear, and after a few hours, a slight purulent discharge was noticed running from the auditory canal. This continued until one week ago, when the discharge became very scant. During this time he had lost considerable in weight, was fretful, slept irregularly, and would often refuse to nurse.

Examination showed a scant, dark colored, offensive discharge in the canal. There was a large perforation of the inferior segment of the tympanic membrane, and pressure over the tissues behind the auricle produced evidences of considerable pain. The temperature at this time was normal, but the condition of the ear, and the general asthenic state of the child very clearly indicated a serious mastoid lesion, and immediate operation was advised. Consent for operation was not, however, obtained at this time, owing to the difference of opinion among the medical advisers.

On the following day the temperature in the morning was 104.8° F., pulse 130, respiration 32; while five hours later the temperature was 99° F., pulse 100 but weak, while the respirations were 28. At the same time, the child looked very much worse, while the auricle was beginning to project, and the mastoid region to become swollen.

On account of the rapid temperature change, and the apparent septic condition of the child, it was considered that the infection had involved the lateral sinus, and the parents were again informed that immediate operation was imperative. This was performed at the same time under ether anesthesia, with the child in an exceedingly serious condition, the temperature having risen just previous to operation to 104.3° F. After making the skin incision and opening the cortex, it was found that the latter was exceedingly thin over the location of the antrum and quite dark in color, while the slightest pressure with a sharp spoon was sufficient to break it down, as in a child of this age the antrum is almost immediately under the surface of the bone. The antrum was found full of granulation tissue, and disorganized bone debris, while the mastoid in great part was considerably necrosed, and softened. This tissue with a small amount of greenish offensive pus was then removed, and the entire mastoid cortex was cut away, and as the aditus contained a large amount of granulation tissue, the upper posterior canal wall was in part removed.

After removing all of the cortex, the carious osseous tissue over the sigmoid sinus was carefully taken away, thus exposing the sinus at the bottom of the bony cavity. On account of the extensive necrosis here, a considerable area of the sinus was readily exposed, but higher up and also lower down to the jugular bulb, the bone was removed with rongeur forceps, so that as large a space was obtained as possible in the necessarily restricted field. On palpating the sinus no pulsation was felt, so it was opened and an aseptic clot was found beginning to undergo disintegration. With the curette, the clot was removed in small pieces, and considerable difficulty was found in obtaining the blood flow from above, but this was finally accomplished, and while the bleeding was controlled by pressure here, little difficulty was experienced in removing the portion of the clot lower down, and a free passage was readily secured. As the child was in a very bad condition, hypodermoclysis was employed, and after rapidly removing all the necrosed bone, the wound was dressed in the usual manner.

For the first two days following the operation, the heart was weak and irregular, the pulse was between 130 and 160, while the temperature varied from 97.5° F. to 102° F. Then the condition greatly improved until the fifth day, when the temperature suddenly rose to 104.5° F. The mastoid was reopened, and a few drops of pus were found in close contact with the sinus. This was removed, a fresh dressing applied, and the case rapidly progressed until, within a few weeks, the child had regained his lost weight, the mastoid wound healing within seven weeks.

Comment. Of the various symptoms more or less indicative of involvement of the sigmoid sinus, rigors, followed by a rapid rise of temperature to 104° F. or 105° F. or more, is one of the most characteristic, but in the very young child it is practically impossible to obtain any evidence of a definite chill, and considerable dependence must be placed, when the mastoid is involved, upon the frequent vacillations of the temperature over several degrees. The pulse rate is often fast, but later it becomes small and weak, and usually offers but little information. When the suppurative changes are extensive, and, as in this instance, have lasted for a considerable time, the skin may show the evidences of the general pyemic condition. It is dry, may be more or less sallow, or even of a decided yellow hue, and the accompanying disturbances of the alimentary canal, such as a heavily coated tongue, and constipation alternating with diarrhea are also present. Vomiting, optic neuritis, and some retraction of the

cervical region are of some value in aiding the diagnosis in adults, in conjunction with other symptoms; but in the infant, the two former symptoms cannot be considered, while the latter is more indicative of some meningeal involvement than otherwise.

Should the thrombus involve adjacent venous channels in its extension from the sigmoid sinus, it is possible to find more or less characteristic local symptoms, which will present features of considerable value in overcoming the diagnostic difficulty, such as edema over the region behind the auricle, possibly extending to the temporal region, when the venous channels in these parts are obstructed; while should the thrombus extend down through the channel of the internal jugular vein, there will develop an inflammatory band like prominence along the course, and the cervical region on the same side will become quite painful to pressure.

All of these symptoms, however, develop so late, or individually are of so little importance in the very young child, that the most reliable diagnostic symptoms of the sinus involvement, during the course of the suppurative otitis media, is undoubtedly the rapid exaggerated oscillations in the temperature range, so that it is highly important in such cases that the temperature be taken at least every two or three hours, both by day and night, in order that such changes are not allowed to continue without being recognized.

DISCUSSION.

DR. EMIL MAYER, New York, N. Y.: When Dr. Oppenheimer asked me to discuss his paper, he also did me the honor of sending me his case histories, so that I could look them over, and so perhaps I am in a better position to judge the whole of the paper on that account. It is almost impossible to discuss in a critical way a paper that covers the ground so fully. You will admit that a man who has studied every single point, including the blood examination, and has reached conclusions that are definite, lends himself very little to anything of a critical nature. I would, however, throw out this suggestion, that as the essayist has large hospitals where he can follow his cases, I think it might be very important hereafter to study the X-ray findings in these cases, and see if it is possible in that way to determine the presence of the thrombus, when it might not otherwise be discovered.

One can only emphasize, then, the importance of examination of the ear discharge. I think we are a little too apt to say we have cleaned out the mastoid and found pus there. We should know the nature of it, so as to be on the watch. The importance of the blood examinations is like the Wassermann test,—when they are positive they tell us a little, when negative they tell us nothing.

The important thing, as I see it, is that as eternal vigilance is the price of liberty, so eternal vigilance in these cases is the price of human life. That Dr. Oppenheimer has been able to save a number of these cases that become hopeless so fast, is one of the greatest compliments that can be made to surgery, and a credit to his ability.

DR. JOSEPH C. BECK, Chicago, Ill.: The number of cases that Dr. Oppenheimer reported of course exceeds any I ever hope to have of this disease. It is an enormous amount of material to report on. He speaks from experience, and it is very valuable to us to have the privilege of listening to his report. I have had a little experience along this line, and, as Dr. Mayer has said, the paper is very complete. We come across cases that differ, and I wish to speak of a different experience I have had. For instance, Dr. Oppenheimer says that he has never observed the Crowe-Beck sign (Beck of Vienna), i. e., the stopping of the jugular vein on one side by pressure gives us not only the choked disc but external evidence of dilatation of even the superficial veins, such as the temporal. A good eye man, looking through an ophthalmoscope, can usually see the resulting papillitis. It is a very good sign. Dr. Suker has examined my cases in the County Hospital with good results, showing that this sign is of value, yet Dr. Oppenheimer has not found it in any of his cases. A condition which he should probably have encountered in many cases, was the absence of discharge from the ear or any evidence of perforation, with other symptoms being present.

Another thing, he never found any other bacteria except the streptococcus. In the last fifteen years or more, I have almost always practiced ligation in symptoms of sinus thrombosis, and I discovered recently a condition of staphylococcemia. That patient was operated upon, though we did not ligate, and made a recovery. I think it should be brought out that the staphylococci are not so virulent. I have another case of bilateral sinus thrombosis. The patient was a little girl who had a bilateral otitis media. We operated on one side; the sinus was thrombosed, and a ligation was done. Three weeks later, she had all the symptoms on the other side, but of course we did not ligate there. That girl had multiple abscesses and loss of an arm, but she lived for about two years, and finally died from the exhaustion of chronic sepsis. Another point which I think of importance is the finding of local symptoms indicative of cerebral involvement.

DR. EDWARD J. BERNSTEIN, Detroit, Mich.: I want to say something which will doubtless strike most of you as rank heresy. We feel that the signs of sinus thrombosis are pretty clearly defined in most cases, and certainly are very distinct when the clot has broken down and the sinus filled with a purulent mass, and that these cases require an operation on the jugular of one sort or another. I am not so sure that even these cases *do invariably* demand ligation of the jugular or exenteration of the clot to free bleeding.

About six years ago, I operated on a mastoid in which, after going down to and uncovering the sinus, a pultaceous mass was found replacing the sinus. For some reason, probably the exhaustion of the patient at the time, I decided to leave the ligation of the jugular till another day,

contenting myself with removing a portion only of the purulent mass. The patient went on to recovery, and did so well that a further operation was declined and not urged. The young man is still alive and well.

About a year or so ago, I was called to see a girl of thirteen with recrudescence of a chronic mastoid, with the old classical symptoms of acute mastoiditis. The girl, though she had a temperature of 103° per mouth, had been to school that afternoon. She was sent to the hospital, and I did a complete radical, exposing the sinus and removing the tip of the mastoid. The sinus wall was bare and open, and its contents were replaced by a large purulent mass. On account of the late hour and the exhausted condition of the girl, I decided to leave the ligation of the jugular till another day. She did very well for two days, then began to complain of pains in various joints. I was greatly alarmed, and said to myself, "Well, you ought to be hung for not completing that operation." However, in six hours the pains disappeared, and the girl made an uneventful recovery.

Now, two cases mean nothing, but Dr. Baum of Denver, Colorado, a short time ago reported four similar cases. It seems likely to me, that the sinus is involved more often than we think, and that a certain number, even in the face of thrombosis or suppuration, will get well without further operation than a complete radical mastoid. Just what types of cases may be so treated I am in no wise able to state.

DR. JOHN F. BARNHILL, Indianapolis, Indiana: Speaking to the last speaker's query as to whether patients will get well, and whether or not the internal jugular should be ligated and resected, I think the experience of every operator, or very many operators, has proven that they do frequently get well without operation; but that does not mean that we are not to be thorough when a patient is in our care, and that we should not ligate because the symptoms are not always clear as to whether or not the pathology is thoroughly walled off in the sinuses or vein. Moreover, we all know that sepsis may be creeping into the system, even though it be walled off and blocked at the main vein. Some years ago, I almost invariably did a resection of the jugular vein when I operated for thrombosis. In one case I recall, in a section of vein from a child, an examination of the resected portion showed the infected clot very beautifully walled off by nature. In this case it was clear that no ligation and no resection could have done better than Nature had already done, but I did not know that, and I am sure that what I did in resecting the vein did not hurt the patient, and therefore, since we cannot always tell whether or not it is walled off, I, for one, think that the few moments spent in resecting or ligating is always well worth while, and adds greatly to the patient's safety.

DR. SEYMOUR OPPENHEIMER, New York, N. Y., (closing): I regret that the limited time prevented the presentation of all the case histories, because they covered quite a variety of cases of atypical sinus thromboses, but they will be published in the Transactions.

Replying to Dr. Bernstein first: I am afraid that he misunderstood me. I did not say that a sinus thrombosis necessarily means that one must ligate the internal jugular. In quite a number of instances it is not necessary to ligate the jugular, and the return flow of blood can be

reestablished without any attack upon the neck portion of the vein, but my belief is growing stronger each day, that I am rather inclined to be an exponent of the primary jugular ligation; judging entirely upon the basis of statistics over a large series of cases, I would be an exponent of primary ligation wherever I can definitely establish my diagnosis.

By way of illustration, I might refer to a case which proves that cases do frequently get along without a ligation of the jugular vein of the neck. Now, what happens in these cases to which Dr. Bernstein refers? The explanation is in the fact that a thrombus extending to the lower end of the vein, still had an aseptic portion at its lower end, the upper end being disintegrated. In the case I refer to, the thrombus extended down in the subclavian and innominate. I split the clavicle and exposed the vein as far as I dared, but knew better than to attempt to remove the great big thrombus which was protruding from the subclavian, because I could not have prevented the entrance of air. I simply split the clavicle and allowed the thrombus to remain. The upper portion broke down, but the lower end was aseptic and inflammatory obliteration of the vessel took place. The patient recovered.

On the question of a double sinus thrombosis requiring double jugular ligation, to which Dr. Beck referred, I have never encountered such a case and hope I never will. I do not know what I would do if I had such a patient. You certainly cannot ligate both jugulars at the same time. I have seen several cases of acute mania and other mental disturbances resulting from the ligation of but one side. I presume if I were confronted with such a case, I would ligate one side and take all the time I could *before* operating on the other, in the hope that some collateral circulation would take place. It does happen, that in some instances the superior petrosal vein is unusually large, and that some return circulation may be established.

The X-ray examination of sinus thrombosis has been rather unsatisfactory in my hands in its interpretation. It has been practiced in a number of cases, but I know of only one satisfactory plate, where the diagnosis of sinus thrombosis was established from the plate before operation.

In regard to the organisms present, I have no doubt that some other organisms might be responsible, but up to the present time I have never encountered in any case of sinus thrombosis any organism other than the streptococcus or the streptococcus mucosus,—that is, in the pus taken from the mastoid process at the time of operation.

REPORT OF TWO CASES OF TUMORS OF THE TRACHEA.

HAL FOSTER, A.M., M.D., F.A.C.S.

KANSAS CITY, MO.

Tumors of the trachea do not occur very often. This reason alone is sufficient for the report of the following two cases. Such tumors are so very important, as to demand our study and attention.

The English, Russian, French, Italian and several of our fellow members, namely Jackson, Cohen, Freudenthal, Law and Ingersoll, all report cases. McKenzie, of London, also reports a number of cases. The majority of these cases occurred in children under ten years of age. All of them were causing alarming symptoms when first seen by the physician. When we consider the size of a child's trachea, from three to five years old, as being only 1 1/2 inches from the cricoid cartilage to the sternum, and in children aged eight to ten about 2 1/4 inches, we will not be surprised at the distressing and alarming symptoms presented in these little patients. Owing to the very sudden onset of obstructive breathing and paroxysms of asphyxia, the diagnosis is very apt to be difficult. The physician is very apt to think of enlarged thymus gland, unsuspected foreign bodies lodged in the air passages, papilloma and even laryngeal diphtheria.

Both of my cases were stricken in the middle of the night with such obstructive breathing, that immediate relief had to be given or death from asphyxiation would have occurred. Relief from the distressing and suffocating symptoms is of prime importance, and now becomes the first duty of the physician. Tracheotomy seems to be required as a life saving procedure, is urgently required and always gives immediate relief, much to the delight of the physician and joy of the parents and friends of the little patient. This operation was done in both of my cases as a life saving procedure, and was preeminently successful. The X-ray should be used as an aid in diagnosing foreign bodies, and the bronchoscope should be used for the same reason when possible.

CASE 1. Boy, age four, was referred by Drs. Gaines, Mann and Phillips, some years ago, from Loan Jack, Mo. Was brought to my office by his father. The boy had been troubled with hoarseness for some time. Very little attention was paid to this

condition, as he was well in every other way. The doctors were called in the middle of the night, one year before I saw patient, as the boy was near death from suffocation. They opened his trachea and inserted a tube, which gave immediate relief, and the life of the boy was saved. Next day, a diligent search was made for evidence of diphtheria, but none could be found. On the third day, the boy was doing so well it was deemed best to remove the tube. On taking out the tube, immediately very severe suffocating symptoms became so alarming, that the tube was at once reinserted, which gave instant relief. After this experience, the patient became so very nervous when the tube was touched, that no other attempt was made to remove the tube. He was in good condition when I saw him and appeared as well as usual. As this was just before the days of the X-ray and bronchoscope, I had only the direct method to go by. He had been wearing the tracheotomy tube over twelve months. He would get excited and exceedingly nervous if the tube was touched, from fear that the tube would be removed and he would be suffocated. I sent him to the Mercy Children's Hospital. Early next morning, I was ably assisted by Dr. Alice Graham, the founder of this hospital. Dr. Seitz administered the ether and Drs. Mann, Gaines and Wilhelm also assisted me. When the patient was etherized, I removed the tube and held the trachea opened with tracheal forceps, and then placed a long sterile feather into the opened trachea to produce coughing, hoping that the act of coughing would expel any foreign body. During the act of coughing, this small tumor, which I now present, was forced up into the opened trachea. As it had a small pedicle, I held it with forceps and clipped it off with the scissors. The tube was reinserted and allowed to remain in the trachea until the next morning. Twenty-four hours later, I removed the tube and kept the boy under personal observation for one week. His recovery was rapid and he never had any further difficulty in breathing. He grew to be a man and served his country in the late war in an honorable way. Dr. Hall, the pathologist, pronounced the growth fibroma.

CASE 2. A number of years later, soon after the X-ray and bronchoscope were discovered, I received a letter from a worthy member of this Academy, Dr. Scales of Hutchinson, in which he said he had a small boy, age six, under his observation, who had been wearing a tracheotomy tube for two years. He would be glad for me to examine the boy at the clinic, and write him the findings before any operation was attempted. Several days later,

the father brought a six year old, healthy looking boy to my office. He appeared well in every way, with the exception he had worn a tracheotomy tube for about two years. When the tube was taken out he would almost immediately suffocate before the tube could be reinserted. I now, in this case, had the aid of the X-ray and bronchoscope. I sent this boy to St. Joseph Hospital, and had my friend Dr. Scott take an X-ray picture to endeavor to locate any unsuspected foreign body that might be lodged in the air passages and had been overlooked, as he had done several times before. He could find none. I wrote to Dr. Scales to come to St. Joseph Hospital and we would look into the boy's larynx and trachea, using the direct method by means of the bronchoscope. Several days later, assisted by Drs. Scales, Lake and Daily at St. Joseph Hospital, under ether anesthesia administered by Dr. Daily, I removed the tracheotomy tube and held the trachea opened with a tracheal dilating forceps and inserted a child's size electric illuminated Jackson's bronchoscope into the trachea. A small tumor could now be distinctly seen in the bronchoscope. As it was held firmly by the long forceps, I clipped it off with the scissors very easily, owing to the fact of its being attached only by a single pedicle. I now removed the dilating tracheal forceps. The tracheotomy tube was reinserted and allowed to remain in the trachea for twenty-four hours. On calling next morning, the boy was doing very well and his breathing was about normal. I removed the tube and never found it necessary to insert it again. I kept him under observation a few days longer. The wound in his trachea united very rapidly; in a week's time he appeared in a normal condition. Six weeks later, he called at my office with his father, on their way to California. Ten years later, they wrote me that the boy was as tall as I was, and had never had any trouble with breathing since the tumor was removed.

It behooves us as physicians, when called upon to treat young children suddenly taken with suffocation, to resort to all the modern methods for making a diagnosis. Dr. Frank Hall, the pathologist pronounced this growth, like the first, a fibroma. The experience I had in the first case aided me greatly in treating the second case. Tracheotomy was badly needed at the time the cases were first seen by the attending physicians, and undoubtedly saved the lives of these children. Both of these boys had been troubled with taking cold and hoarseness for some time. Dyspnea and hoarseness in young children should never be neglected; the cause should be looked for and removed.

DISCUSSION.

DR. RICHMOND MCKINNEY, Memphis, Tenn.: I am greatly pleased that Dr. Foster did not report a dozen cases of this condition, for in many years' practice I have seen only one case of intratracheal tumor. It is rather a rarity. I had a good opportunity in December to see a case in a sixteen-months-old child, who had quite a different history from Dr. Foster's cases, for there was no suddenness of onset. This child had a croupy cough and hoarseness, and had been treated with X-rays for an enlarged thymus gland for several months. I found, on passing the bronchoscope, a smooth and hard growth beneath the left vocal cord. I thought at first it might be a papilloma, but it did not have the appearance of that kind of a tumor. I took it out without anesthesia, which always adds greatly to the risk in these cases. I removed it in two sittings with the use of the direct speculum, and the Bruenings' biting forceps. I was surprised to find it was a fibroma, for the child was very young for the occurrence of a growth of that nature. There was no return of the growth, as there usually is in papilloma.

It is interesting to hear of cases of this kind. This is the only case I have seen in a considerable bronchoscopic experience. Papilloma is rather common, but it cannot be called an intratracheal growth. The case I report, and the ones Dr. Foster reports, were true intratracheal tumors. There is little discussion to be had on these cases, but I wish to point out the very great ease of their removal by the direct method, and to suggest that you do not use ether if you can possibly avoid it, and you can avoid it by using a simple technic of the sort described.

DR. EMIL MAYER, New York, N. Y.: I wish to add the report of another case, in this brief study of three cases of fibroma of the trachea. In a child of nine, who had the same progressive symptoms Dr. McKinney described, and where the diagnosis was made by the laryngoscope and the tracheoscope, a tumor about twice the size of the one shown by the reader of this paper was removed, and proved to be a fibroma. I think we should call attention to the fact of our individual experiences when these occur, and we should bear in mind that there is such a thing as a fibroma of the trachea.

DR. ROSS HALL SKILLERN, Philadelphia, Pa.: This is a unique pathologic experience, and I think if any of us have had any experience along this line, it would be very valuable if we would report it briefly at this time.

DR. J. M. INGERSOLL, Cleveland, Ohio: In 1914, I reported to the American Laryngological Association a case of primary sarcoma of the trachea, which was the first one I had ever seen. At that time, only ninety-four cases had been reported in the medical literature. Dr. Chevalier Jackson, in discussing the paper, spoke of the rarity of primary cancer of the trachea, and said that he had seen only one case. Extension of these growths from the larynx into the trachea is, of course, not unusual, but primary tumors of the trachea, whether benign or malignant, are comparatively rare. Dr. Foster has been very fortunate in seeing several of these rare cases, and his success in treating them is very interesting.

DR. EDWARD J. BROWN, Minneapolis, Minn.: In 1909, a young man came to me (D. K. 18), complaining that he had had difficult breathing since he could remember, and had worn a tracheal tube for one week, for diphtheria at the age of four. Four months before coming to me, his very large and obstructing middle turbinates had been removed by a competent surgeon in Chicago, at which time there was no evidence of sinusitis. No examination of the trachea or larynx had been made. I found pus in both nares, crusts in the right, tissues covering the right unciform process swollen, and the right frontal sinus more sensitive to pressure than the left. The stethoscope over the tracheal scar revealed an exaggerated expiratory roughness at that point. The laryngoscope revealed nothing. On the second morning, when he was to have come for further investigation and possible operation, his physician telephoned that he had died suddenly in the night. No autopsy could be secured.

DR. GEORGE W. BOOT, Chicago, Illinois: Some years ago I had the pleasure of assisting Dr. Beck with a tumor of the trachea. The growth was about the size of a small marble, and was a carcinoma secondary to a carcinoma of the thyroid.

DR. JOHN F. BARNHILL, Indianapolis, Indiana: I have no report to make in this line, but would like to ask Dr. Foster what his opinion is concerning the value of the tracheotomy where the obstruction lies below the seat of the tracheotomy, as in his case. It would seem that in such obstruction as described, tracheotomy could by no possibility be of help.

DR. HAL FOSTER, Kansas City, Mo. (closing): I have very little to say. I have seen only the two cases. Dr. Ingersoll has seen one, and Dr. McKinney reports one. He perhaps did not understand what I stated. The onset was not sudden; the children had been complaining for some little time, but the parents did not pay any attention to them as the symptoms were slight. They would be a little hoarse, and finally the growth got so large it gave them trouble, and that symptom came on suddenly, during the night.

I would not do a tracheotomy now. I would try the operation without ether now, but the tracheotomy gave relief. I saw the child almost suffocated after the tube was removed. We were called to relieve these children during the night. The moment I took out the tube the little tumor fell right into the opening, and I had no difficulty in removing it.

Both these growths were fibromas and not granulomas. Granuloma is caused by allowing the tube to remain a long time after tracheotomy, and never occurs before tracheotomy.

DR. JOSEPH C. BECK, Chicago, Illinois: I wish to speak about the point Dr. Foster just mentioned, which is important. These are old cases of tracheotomy. Everyone knows that granulations form around the cannula, and it is not at all unlikely that these tumors could be granulomas which have been caused by wearing the tracheotomy tubes. I have removed such growths but, of course, they cannot be called tumors, but simply granulomas. The question is what was the condition of the cords? Jackson and others say, that in many instances, as a result of long continued inactivity, the vocal cords fail to open, and when the tube is removed the walls fall in and the patient chokes. Therefore, a closer observation of the larynx at the time of operation, to determine whether or not it is functioning, is very important.

OTITIS MEDIA COMPLICATING OPERATIONS ON THE GASSERIAN GANGLION.

HORACE R. LYONS, M.D.

ROCHESTER, MINNESOTA.

Otitis media complicating operations on the gasserian ganglion was first observed in the Mayo Clinic about three years ago, in a patient who complained of a sense of fullness and deafness following section of the posterior root of the gasserian ganglion for trifacial neuralgia. Since then the observation has been made several times. This type of otitis media occurs from a few days to several weeks after the gasserian ganglion operation. The chief complaint is a sense of fullness and deafness on the side on which the operation is performed. Otalgia is never severe and is not a usual complaint. Examination of these ears discloses a fullness in the inferior quadrants of the tympanic membrane, with more or less obliteration of the common landmarks. There is usually a fluid level, and a bubbling sound is heard with inflation of the eustachian tube. Two patients said that they had "water in the ear." The tympanic membranes were pale in each instance. There also appear within the middle ear bleb like formations, without evidence that the rest of the cavity contains fluid. The tympanic membrane is gray or pink and never shows generalized fluid. It is flaccid or atrophic in appearance, never intensely red, and never has the sense of resistance with the otoscope. Deafness, as tested by tuning forks, was of the conduction type and not severe in degree. Drooping of the posterior superior canal wall was not present.

In one case (Case 1), secretory otitis media followed alcohol injection of the posterior root of the ganglion. Spontaneous rupture and secondary infection, resulting in suppurative otitis media, occurred. It is also of interest that the posterior root was injected, since the neuralgia disappeared at once and cutaneous anesthesia followed.

In another case of secretory otitis media, not reported because the condition occurred several weeks after the patient went home, and our information was not definite, suppuration occurred, but cleared up promptly. This man had a corneal ulcer, as did one other patient.

Abrasions of the cornea are not uncommon following operations on the gasserian ganglion; two of this series of patients had corneal ulcers, but I am unable to determine whether such ulcers are due to trauma or purely to trophic, local changes. Both causes may be common, and possibly a combination of the two, namely lowered local resistance due to the trophic changes, with subsequent trauma from dust and other foreign materials. Although goggles of the automobile type are worn by such patients postoperatively, trauma cannot be ruled out definitely.

In many respects, the picture of otitis media due to trophic disturbances following gasserian ganglion operations, simulates herpes zoster oticus, differing chiefly, however, in that pain is absent in the former and severe in herpes zoster; also the bleb formations on the pinna are absent in this form of otitis media.

NERVE CONNECTIONS.

Sensory and trophic nerve supply to the mucous membrane of the middle ear is abundant and is directly connected with the sensory root of the gasserian ganglion. The tympanic plexus in the middle ear also has direct connection with the posterior root of the fifth cranial nerve, the sphenopalatine ganglion, and the cervical sympathetic nerves.

The sensory nerves to the external auditory canal are derived from the auriculotemporal branch of the trigeminus, and from the auricular branch of the pneumogastric. The latter, also known as Arnold's nerve, perforates the wall of the meatus and supplies its lining membrane.

The posterior wall is separated from the mastoid process by the tympanomastoid fissure. The auricular branch of the pneumogastric nerve passes through this fissure to the posterior wall of the canal. The coughing, sneezing or vomiting that sometimes follows irritation of the canal, as from cleaning the ear, or examining it with instruments, is said to be due to a reflex effect on the pneumogastric through this branch. The auriculotemporal branch of the trigeminus nerve enters into its supply, and may explain the earache in cases of cancer of the tongue or disease of the lower teeth.

The nerve supply of the tympanic membrane is derived chiefly from the auriculotemporal branch, supplemented by twigs from the tympanic plexus and by the auricular branch of the vagus. They accompany, for the most part, the blood vessels and, besides supplying the latter, form a subcutaneous and a submucous plexus. The tensor tympani and tensor palati muscles

receive their nerve supply from the same source, namely, the trigeminus, through the otic ganglion.

The nerves supplying the mucous membrane of the tympanum are branches from the tympanic plexus formed by the tympanic branch of the glossopharyngeal nerve, in conjunction with sympathetic filaments from the network accompanying the carotid artery. The tensor tympani muscle receives its supply from the trigeminus, the stapedius from the facial. Although the chorda tympani nerve has an intimate topographic relation to the space which it traverses close to the outer wall, it gives no filaments to the structures within the tympanum. The nerves of the eustachian tube are supplied from the tympanic plexus, and from the pharyngeal branches from the sphenopalatine ganglion.

The tympanic nerve, or Jacobson's nerve, arises from the petrous ganglion and traverses a tiny canal in the osseous bridge between the jugular fossa and the carotid canal. Entering the tympanic cavity and receiving fibers from the carotid plexus of the sympathetic by way of the small deep petrosal, the tympanic nerve passes upward and forward in a groove on the promontory and breaks up in this situation to form the tympanic plexus. After distributing filaments to the mucous membrane lining the tympanic cavity and the associated air spaces (mastoid cells and eustachian tube), its fibers reassemble and join with a filament from the geniculate ganglion, to continue as the small superficial petrosal nerve to the otic ganglion.

The branches of the tympanic nerve are: the small superficial petrosal nerve, the branch to the fenestra ovalis, the branch to the fenestra rotunda, the branch to the eustachian tube, the branch to the mastoid cells, and the branch to the great superficial petrosal nerve.

The auricular branch of the vagus is given off from the ganglion of the root. It receives a filament of communication from the petrous ganglion of the ninth nerve, and follows the outer margin of the jugular foramen to an opening between the stylo-mastoid and jugular foramina. Entering this foramen, it traverses a canal in the temporal bone, which crosses the inner side of the facial canal and terminates between the mastoid process and the external auditory meatus. Leaving the canal, the nerve supplies the skin of the posterior part of the auricle and of the posterior inferior portion of the external auditory meatus. While traversing the temporal bone, the auricular nerve communicates with the facial and, after reaching its area of distribution, with the posterior auricular nerve.

The communicating branch of the facial nerve to the tympanic plexus traverses a tiny canal in the temporal bone to reach the tympanic cavity, where it joins the main continuation of the tympanic plexus of the glossopharyngeal to form the small superficial petrosal, and proceeds to the otic ganglion, which it enters as a sensory root. The fibers from the tympanic plexus, probably secretory in function, are distributed from the otic ganglion to the parotid gland.

REPORT OF CASES.

CASE 1. (A398947) Mr. S. A. H., aged forty-eight years, came to the Clinic complaining of severe pain throughout the left side of his face, which was recognized as trifacial neuralgia. July 28, 1922, the patient was relieved by a preliminary alcohol injection of the sensory root of the fifth cranial nerve. He returned in about two weeks, because he had a recurrence of the severe pain. Examination of his ears was negative. August 10, a gasserian ganglion operation was performed on the left side, the posterior root being cut. Three days after operation, the patient complained that his left ear felt full; this persisted, with slight deafness.

Examination showed the right ear to be normal, the left tympanic membrane was pale, the lower quadrants were bulging, and the upper ones were normal. Motion of the tympanic membrane with an otoscope confirmed the patient's suspicion that there was water in his ear, because the fluid could be seen to move. Changing the position of the head changed the relation of the fluid level to the ear landmarks. Eustachian tube inflation caused a bubbling sound. Functional examination of the hearing revealed a slight conduction type of deafness. The patient had no otalgia at any time. Paracentesis was not necessary and the patient was dismissed three days later with his ear objectively and subjectively normal. In a letter, August 18, he says that symptoms of fullness and deafness are decreasing. The fluid level is lower.

CASE 2. (A359235) Mr. W. B., aged forty-eight years, had typical trifacial neuralgia. His ears were normal. Three alcohol injections were given on the right side, May 28, December 15, and December 19, 1921, respectively. The last injection afforded relief, but the patient had a sense of fullness and deafness in the right ear. The ear ruptured spontaneously, and a thin watery fluid escaped. No further treatment was necessary. December 29, 1921, a gasserian ganglion operation was performed, the right sensory root being sectioned. Three days later, before

the primary dressing was changed, the patient complained of a slight pain in the right ear, soon followed by a spontaneous discharge of clear fluid.

Examination revealed a pale tympanic membrane, slightly full in the lower quadrants, with a posterior inferior perforation, and a thin fluid pulsating through the opening. This clear fluid became definitely purulent on the following day by secondary infection. Slow but steady improvement then occurred. January 14, 1922, when the patient was dismissed from observation, the tympanic membrane was retracted, the perforation healed, and subjective and objective symptoms were entirely relieved.

CASE 3. (A365379) F. N. B., aged sixty-five years, had trifacial neuralgia. Preoperative examinations revealed normal ears. July 30, 1921, the sensory root of the gasserian ganglion was sectioned. About nine days later, the patient complained of fullness and slight deafness in the right ear.

The typical picture of a pale membrane with slight fullness in the lower quadrants and the slight deafness typical of the condition were present. There was no otalgia; paracentesis was not considered necessary. The patient was seen daily and the ear rapidly cleared. This patient also had a corneal ulcer in the right eye, which was typical in its resistance to local treatment. Further trauma was eliminated by the use of a glass eye shield.

CASE 4. (A358935) Mr. T. E. G., aged sixty-eight years, had a right gasserian ganglion operation for trifacial neuralgia, May 19, 1921. The sensory root was cut. The ears were found to be negative. The patient was dismissed from observation without complications. However, a letter from his home physician revealed that he had subsequently developed a right otitis media, which spontaneously ruptured and became secondarily infected. He also developed a right corneal ulcer. Both resisted local treatment. A later letter, December 19, 1921, stated that both the ear and the eye were about cured.

Four other cases belong to this series, but since a personal examination was not made, they are not incorporated in this report. The patient in Case 1 was typical in every detail and most interesting. Parenthetically it must be said, however, that all the patients had complete relief from the pain after operation; they also had the cutaneous anesthesia over the surface supplied by the posterior root of the ganglion.

It should also be noted that two of the four patients had trophic disturbances in the form of a secretory otitis media and corneal ulcer; this again brings up the question of the factor of trauma in these cases of postoperative corneal ulcers.

SUMMARY.

1. Secretory otitis media following gasserian ganglia operations is an entity. It is due to trophic disturbances in the mucous membrane of the middle ear.
2. The nerve connections between the gasserian ganglion and the mucous membrane of the middle ear are abundant.
3. The ear and eye complications are always on the side on which the operation is performed.
4. Trauma, as an etiologic factor, is eliminated so far as the ear is concerned, and this gives further basis to the argument that the corneal complications are entirely trophic in origin.

DISCUSSION.

DR. IRA FRANK, Chicago, Illinois: The condition described is an interesting after effect, following section of the posterior root of the Gasserian ganglion. That operation and condition bear a direct relationship to each other, appears to be established by the occurrence of the ear involvement always on the side of the operation, and within a few days of the latter. Apparently, it is not frequent enough or serious enough to constitute a contraindication against operation when the latter is necessary. But its occurrence, having been pointed out, the possibility of its development should be borne in mind, in order that the development of ear symptoms may be properly interpreted and treated.

While the causal relation of lesion and operative procedure is granted, we doubt whether sufficient proof has been adduced to place the lesion in the group of trophic disturbances. The latter, in general, occur in analgesic areas, and trauma is usually a factor which cannot be excluded, and which may be as important as the loss of supposed trophic nerve fibers. It is difficult to conceive that the cessation of nerve impulses can cause changes other than an atrophy of slow progression. In the middle ear, where local trauma can not be a factor, one would expect a trophic effect following nerve section to manifest itself by atrophic changes rather than by secretion or exudation. The latter changes are such as one would expect to follow nerve stimulation. The early occurrence of the lesion described, also speaks against a trophic change. It would appear more probable that the condition is the result of stimulation, by the trauma of resection and the consequent reaction of vasodilator fibers. Stimulation of the latter, followed by vascular dilatation, might lead to exudation of fluid. When alcohol is used, there might also be a period of stimulating action; or it is possible that some of the alcohol may have entered the Eustachian tube, the ear condition being then the result of local irritation rather than of trophic disturbance.

Some objection may also be raised to the use of the term "secretory" as applied to the condition. As indicated above, it is not believed that increased secretion ought to occur as the result of trophic changes. If the accumulation of fluid is the result of exudation following vascular dilatation, which appears to be the most probable explanation, then it

might be better to term the interesting condition described as an acute serous or exudative otitis media. Against the use of the term otitis there may be some objection, since the reaction is probably not an inflammatory one, if its causation is either trophic or due to vasodilatation.

DR. JOHN F. BARNHILL, Indianapolis, Indiana: The subject of dealing surgically with the Gasserian ganglion is rather new, and therefore a great many questions have arisen that are not as yet well understood. Frazier has pointed out that interference with the first division of the trigeminal nerve is more apt to give rise to disturbances of the eye, than is injury to any other portion, and hence he advises in so far as possible to avoid operative interference with that division. The cases here reported are the first describing middle ear difficulty as a result of ganglion operations, and certainly are not easy to explain. I have never seen any ear trouble following ganglion operation. Several years ago, I began injecting the ganglion with alcohol and have probably so treated in excess of seventy-five cases. In none of these injected cases have I seen ear trouble except pain, when it seemed probable the injection was by error of technic, into the Eustachian tube. I have, however, seen other inexplicable results. Among these have been complete facial palsy, partial facial palsy, and trophic disturbances of the eye. Occasionally, severe pain follows unsuccessful injection of the ganglion with alcohol, which probably is due to the alcohol entering the Eustachian tube. I have done twelve resections of the posterior root. Numerous cases have been reported in which facial palsy has followed division of the posterior root. I had made eleven sections of this root without any such disturbance, and then the twelfth case, done recently, had complete facial palsy following resection of this root. Now, how are these occurrences to be explained? The explanation which the essayist gave of the various nerve rootlets and sympathetic branches, and the very exhaustive description of the nerves supplying the ear, do not wholly explain the surgical misfortunes that sometimes occur subsequent to ganglion injection or root resection. For instance, how is it possible for an injection of alcohol into the Gasserian ganglion to cause a complete facial palsy? How is it possible for a section of the posterior root to cause complete facial palsy? Yet palsy from these causes occurs and has been reported by several operators. We are led to the conclusion that there must be a relation between the Gasserian ganglion and other of the cranial nerves which has not been clearly demonstrated and is not yet understood, for otherwise, facial palsy, eye lesion and the ear disturbances described in the paper would not occur following operations on the Gasserian ganglion. They certainly are not symptoms resulting from traumatism of the brain or ganglion itself.

I am inclined to think that one of the cases of ear trouble that the doctor has reported may have resulted from injecting the alcohol into the Eustachian tube. I infer this from the fact, as reported, that the injection was not a success. The alcohol must have gone some place adjacent to the foramen ovale, as I felt sure it did in one of my cases, for about the easiest place, an error easy to make, is to inject the alcohol into the Eustachian tube. There is reason to believe this may have happened because a ganglion operation was necessary the following day, the essayist reports, and if the alcohol had gone into the

ganglion it would not have been necessary to do the major operation so soon after the injection.

DR. W. LIKELY SIMPSON, Memphis, Tenn.: This is certainly very interesting, but whether or not it is a middle ear infection I am not here to say. I want to take about a minute to report a case with some rather interesting phenomena, a case which is very similar to some of his.

The patient was a woman about forty years old, who had had typical trifacial neuralgia about four years before she came under my care. On July 2, 1921, a neurologist made an alcohol injection into the ganglion, with some relief of pain, and again on July 17, 1921, an alcohol injection was given. Following this injection, there was paralysis of the seventh nerve, with good relief from pain. There was a slight watery discharge from the canal, with slight redness of the drum. The hearing was quite impaired. On July 30, 1921, because of pain in the face and so forth, the radical Gasserian operation was done. I saw this patient previous to the injection, and the ears were normal, as were the nose and throat. There was no cold or infection about the nose and throat following the injection, so far as I could make out, but within the next day or two, there was a mild type of middle ear infection, with the watery discharge. The opening in the drum was very small, if it could be seen at all. Following the radical operation there was no pain, but the cornea was rough and some of the epithelium was lost, so it was necessary to sew the lids over the cornea. The infection in the middle ear continued for about two or three weeks. On August 26, 1921, the ear was dry and tuning fork tests were normal, although the patient claimed that she could not hear at all. She proved to be a malingerer, and the hearing actually was twenty feet for the whispered voice.

This history seems to bear out Dr. Lyons' ideas that this condition is probably a trophic change, as there was not an active suppurative otitis media at any time.

DR. JOSEPH C. BECK, Chicago, Illinois: I can see nothing mysterious about the formation of this fluid in the ear, following operation or injection. The question as to whether the cutting of the posterior root is concerned with this is the point to consider. If the secretory fibers are left intact, as at Rochester and in Frazier's clinic where I have seen it, I say that we can have cessation of the symptoms and stimulation of the secretion. I have had no ear symptoms in my work, but have had one very serious eye complication. It seems to me that in such an institution as that at Rochester, they would attempt to prove this point by animal experimentation. It appears to me, in the cases that have been described by Politzer, Swartze, and others as otitis media and earache, that these symptoms do not occur in operation on that side. I think it would be well to see if they cannot produce this by animal experimentation. I have never read anything on this subject, and think there is nothing in the literature anywhere.

DR. FREDERICK T. CLARK, Westfield, Mass.: In connection with Dr. Lyons' very interesting paper, and Dr. Barnhill's comments regarding the complications that may occur following severance of the Gasserian ganglion, I might mention a case that came under my observation, that

was operated upon by one of the famous neurologic surgeons of Boston. The patient was a woman of perhaps seventy years, who had had a typical trifacial neuralgia. She was operated upon, and within two months after returning home, she developed what I assumed to be an otitis media, without much pain. The membrana tympani melted away and the ear discharged, but after a few days the condition cleared up. Within eight or ten months following that, the patient began to develop trophic disturbances of the eye, as mentioned by Dr. Barnhill, which went on to complete destruction of the vision, and the eye had to be enucleated. The lesion was a chronic keratoiritis. In addition to this, she has since developed difficulty in swallowing, and I am frequently summoned to give her relief at night—simply to relieve her dysphagia. She has the difficulty in swallowing which Dr. Barnhill has mentioned.

Dr. H. R. LYONS, Rochester, Minn., (closing): Because of the fact that nothing has been found in the literature, I went into the case pretty thoroughly with neurologists, regarding the nervous anatomy, and whether we should call this a secretory or serous otitis media. I think we could raise the same objection to both of these terms. I think, from the neurologic standpoint, it is recognized that trophic disturbances follow resection of the ganglion. So far as the ear is concerned, it is not proved unless we can consider a transitory exudation as the preliminary symptom. The whole clinical picture would fit very well with a trophic disturbance. The corneal ulcers have for a long time been recognized as a complication of this type of operation, and it has been fairly well determined, at least by men writing about this subject, that it is a trophic disturbance. Here, again, some symptoms come on immediately and some develop weeks or months later, of the same type, and respond poorly or not at all to treatment. Again, the time limit is not constant, so if we would expect the trophic disturbance to come on late, we have the same thing in the eye that comes on early. The exact cause is much in doubt, but a trophic disturbance at the bottom of it is as good an explanation as can be had. I am much interested in Dr. Barnhill's discussion. I have seen a few cases of facial palsy following this type of operation, and in each case it has been noted that there is a possibility, or it is definitely stated that one or the other of the two other roots were damaged, or cut, or compressed at the time of operation. So in the few cases I know about, there has been definite injury of the other roots of the ganglia. In regard to the alcohol injection, in the particular case I referred to we had the type of ear disease, following the injection, that has been mentioned. You remember it was the third injection that gave relief for two weeks, and following this injection, and not the other two, the ear complication arose. This was done at the Clinic.

The case that Dr. Simpson reported is apparently of the same type, and is very interesting.

In regard to Dr. Beck's discussion, it seems that a good deal could be learned by doing some animal experimentation in the way of resecting the posterior root, or its ganglion, and having the animal under complete control. It is an interesting suggestion, which was considered but given up, because in the experimental work the ganglion is not involved in any pathology.

SOME CASES OF PREHISTORIC NASAL PATHOLOGY.

FRANK A. BURTON, M.D.

DENVER, COLO.

At the present time, considerable interest is being taken in paleopathology, and for several years I have been especially interested in the study of the head from this viewpoint.

My first paper connected with this subject was on "Pre-historic Trephining of the Frontal Sinus", read in 1920 before the Eye, Ear, Nose and Throat Section of the California State Medical Society. My candidates thesis for fellowship in the Triological Society on "Aural Exostoses" dealt largely with this diseased condition in prehistoric man. As far as I know, there is practically nothing in the literature on nasal paleopathology. In the Science of Man Building of the San Diego Museum, there is a large number of unclassified and unreported specimens of paleopathology of the nose. The skulls of the collection were selected by Dr. A. Hrdlicka of the Smithsonian Institute from four thousand eight hundred Peruvian crania.

The purpose of the present paper is to record some observations as to the rhinologic pathology found, as well as to show lantern slides of characteristic specimens. In reporting the results of the study of these dry bones, one assumes that the imagination may be allowed full freedom. But the writer will attempt to interpret the prehistoric pathology under consideration, in terms of our knowledge of present day pathology. My initial purpose was to add light, if possible, to the question of the etiology of septal deviation, through investigation of prehistoric crania. As the work progressed, other features were noticed that seemed to warrant reporting as a contribution to scientific data, together with evidence that nasal pathology is not a condition characteristic of modern man only.

Did the prehistoric inhabitants of Peru have trouble with their antra? I believe they did, but they were not so fortunate as to have the professional care of a member of the Academy. What was done to them is still being done to our lay brothers of the present. Yes, I use the term "was done to them." I recognize the fact that during recent years con-

siderable progress has been made in dental science, and while I do not desire to be harsh on the modern up-to-date surgeon dentist, I must say that many of the dental profession still follow the example of their ancient prototypes of the Andes. Two skulls showed healed fistulae through the second bicuspid sockets into the antra, and one through the first molar



Fig. 1. Fistula into antrum.

(Fig. 1). Whether the tooth dropped out from the abscess cavity or was pulled out because of decay and pain, I cannot say, but the latter seems more logical, as there is evidence that the ancient Peruvians possessed the ability of observation and analysis. We can easily imagine the clinical picture of pain about a diseased tooth spreading up into the face. The patient may have endured his suffering until a full blown infec-



Fig. 2. Fistula through into antrum. Tooth apex in cavity.

tion of the antrum developed, and for relief of the "devil" in his face "trephined" or pulled the tooth with a resulting free flow of pus and diminution of the symptoms. Irrigation of the antrum was probably not performed, but the idea of treating an infected antrum through the alveolar process seems to have originated long before Columbus started West. One skull, as you will see on the screen, shows very clearly the connection between apical abscess and antral infection. (Fig. 2.) The abscess cavity surrounds the apex of the tooth and communicates through into the antrum and also into the mouth. It is only natural to suppose that the antra in these cases were infected, judging from what we know of present conditions. No doubt in a large percent of the cases of tooth, antral, and frontal abscess, relief was sought through trepanation. This was due to the ancient theory of disease; that all pain was from a confined evil spirit trying to regain his freedom. It is a known fact that considerable skill was acquired by the ancient Peruvian surgeons in the use of the flint and obsidian trephine. A surgeon of the stone age, with his meager knowledge, could be excused for trephining directly into the painful area, but I cannot understand why the modern surgeon dentist should treat empyema of the antrum through the alveolar process or canine fossa, thus exposing the sinus to the mouth flora, and endangering his patient with the probability of a permanent alveolar or canine fistula. Yet dentists are doing this very thing in a multitude of cases today, and in many instances producing permanent fistulae. But all the antrum trouble did not result from infected teeth. One skull shows a healed wound in the anterior wall (Fig. 3.) The edge of the perforation is depressed and smooth as though made by a stone instrument or weapon. You will see this on the screen also. It is interesting to note that Moodie reports a similar condition in the skull of a Pleistocene musk ox. A large fistula extends into the maxillary sinus, resulting probably from a blow in a fight with some horned animal. Two other specimens were found, that on superficial inspection seemed to show pathology connected with the antrum; however, close examination revealed the error. Connecting the mouth with the right nasal cavity anteriorly, were two openings through the hard palate, one 13 mm. in diameter, and the other 5 mm., edges smooth (Fig. 4). The antrum was very small and lay laterally to the fistulae. The other case presented a healed sinus from the right canine socket into the floor of the nose, 5 mm. in diameter (Fig. 5). One may speculate as to the causes.



Fig. 3. Perforation left maxillary cavity.



Fig. 4. Fistula into antrum and nasal cavity.



Fig. 5. Fistula into nasal cavity.



Fig. 6. Erosion of frontal floor and bony new growth.



Fig. 7. Erosion of floor and distention of wall of frontal sinus.



Fig. 8. Nasal fracture. Anterior view.



Fig. 9. Nasal Fracture. Lateral view.



Fig. 10. Hypertrophied middle turbinate. Deviated septum.



Fig. 11. Cystic middle turbinates.



Fig. 12. Displacement of choanae.



Fig. 13. Artificial deformation. Lateral view.



Fig. 14. Artificial deformation. Posterior view.



Fig. 15. Extreme septal deviation.

In addition to the cases of prehistoric frontal pathology, reported in the paper on trephining, I have found two more cases of frontal involvement. In one (Fig 6), the orbital floor of the left sinus is gone and the inner wall is studded with small bony excrescences, cauliflower like in appearance. In the other (Fig. 7), part of the right floor is gone and what remains bulges into the orbit, the inner walls being smooth. It is as though a mucocele or pyocele existed, gradually causing a distention of the paper thin floor, a condition whose existence some deny at the present.

Due to the well known combative tendencies of primitive peoples and the less refined methods of warfare, fractures were probably more common than at present. The swinging of the heavy stone weapons indiscriminately at the heads of the enemy probably resulted in a large number of nasal fractures, one of which is especially interesting, as you will see. These fractures all healed without correction of the displacement. Six instances in all of this type of injury were found in the collection, and most interesting were two in which the force acted directly against the nose driving the fragments inward, which in one case left the anterior nares but 1 cm. in diameter. The septum apparently shared in the injury in two skulls, for the perpendicular plate of the ethmoid was displaced and thickened, while in another the olfactory fissure on the left (displacement being to the right) was filled with osseous tissue to the lower level of the nasal bone. (Figs. 8, 9.)

Compensatory middle turbinate hypertrophy accompanied septal deviation then as now, being well marked in four crania (Fig. 10). Hypertrophy was not dependent on deviation however, as one striking illustration shows, which will be thrown on the screen. Here the anterior ends of both middle turbinates showed marked distention due to cell formation (Fig. 11). The right contained a single cavity, 30x7x25 mm., and the left two about half the size of the former. The same pathologic influence to which Sluder and Wright point as the cause of abnormal development in the nose, acted then as it acts now, giving conditions identical through centuries of human development.

While at rare intervals I have noticed differences in the level of the floors of the nasal fossae anteriorly, I do not remember seeing the condition posteriorly, i.e., an asymmetry

of the posterior nares. It may occur and be passed by unnoticed, because of the presence of the soft tissue or mere inattention to a detail which would have little if any pathologic significance. Six skulls present a marked difference in the size the level of the posterior choanae (Fig. 12). Five of these were present in the artificially deformed skulls, or 15%, and one in an undeformed cranium, or 6/10 of 1%. One or other of the parietal bones bulged lateroposteriorly in the deformed skulls, but no relation could be seen between the direction of the bulge and the change in the level and size of the choanae.

As I remarked at the beginning of this paper, the incentive leading to this investigation was a desire, or rather a curiosity, to study the relation between septal deviation and development of the cranial box. It is needless to consume our time retailing the many causes assigned to septal deviation. You all know them, or if you have forgotten you know where to look them up. What I desire to show is that the development of the cranium is a decided factor in the etiology of this condition. Thirty three artificially deformed crania show twenty deviated and eleven normal septa, with two so macerated that all intranasal structures are gone (Figs. 13, 14, 15). This gives a percentage of $60 \frac{2}{3}$; 85% of this number were simple bowing of the septum or bowing plus a ridge, bowing alone being 75% of the cases. 10% of the entire number were ridges and 5% spurs. Of the 152 undeformed crania, 40, or 26%, presented deviations. In this series, a larger number and percent were so macerated that the normal structures are also missing. Making the percent of maceration the same in both classifications, and considering all the additional macerated specimens as deviations, still the figure is only 36%, almost 2 to 1. The percent of bowing and combined bowing and ridges was 70, simple bowing 65%, ridges 22.5% and spurs 7.55. To me the figures indicate that alterations in the development of the size and shape of the cranial cavity play a major role in causing septal deviation, especially simple bowing, or bowing plus a ridge, and that simple ridges or spurs, which are rare, come from other sources than disturbed development of the head.

DISCUSSION

DR. RALPH A. FENTON, Portland, Ore.: I should like to ask Dr. Burton as to the probable incidence of syphilitic conditions in the early Peruvians, to account for the bone changes noted, especially the one surrounded by osteophytes.

DR. GEORGE W. BOOT, Chicago, Illinois: With reference to deflected septum, I wish to state that I have seen a deflected septum in a three months fetus.

DR. FRANK A. BURTON, Denver, Colorado (closing): In attempting to answer Dr. Fenton's question, I should like to say, that although I have been studying this subject for some time, I do not know as much about it as I should like. It should be said that anyone who studies paleopathology will do considerable guessing, and will have opportunity to use his imagination. The men who have studied the evidences of syphilis of the bones have found very different indications than those in any of these cases. The syphilitic specimens in the San Diego Museum have been classified and show characteristic bone changes. None of this series of specimens, that I have presented today, show pathology due to syphilis.

In regard to the statement relating to the fetus of three months having a deflected septum, I have nothing to say except that it is undoubtedly true, and I assume that others could report similar cases. The one point I wish to make is, that in this series, the artificial deformation skulls show a marked increase in frequency of septal deviation over the undeformed skulls. There must be a reason, and to me the explanation is, that the constriction of the skull in these cases of artificial deformation exerted pressure from above downward, and that this force was added to the usual pressure from below upward. By the same process of reasoning, is it not obvious that the increased pressure against the floor of the cranial box, due to the greater development of the frontal lobe at the present time, is a large productive factor in the increase in number of septal deviations?

ABSCESSES DESCENDING FROM THE UPPER AIR
PASSAGES INTO THE NECK AND MEDIASTINUM.
TYPICAL EXTERNAL OPERATION, COMBINED
WITH PROPHYLACTIC MEDIASTINOTOMY.

OTTO GLOGAU, M.D.

NEW YORK CITY.

In the "Festschrift" to Prof. M. Hajek's Sixtieth Anniversary, and in the July, 1922 issue of the Laryngoscope, I have in detail described the Typical External Operation for the Abscesses Descending From The Upper Air Passages. Through the courtesy of my esteemed Teacher, Prof. M. Hajek, I could work last summer at his Clinic as Aspirant, and had thus an opportunity to evolve this method, basing it upon studies made on the cadaver, on the observation of the cases operated upon during my stay at the Clinic and upon the perusal of the records.

I will report to-day three cases which I have operated by this method, since my return to this country, and will point to the preceding ones, including one old case of mine. A brief description of the topographic anatomy of the region involved will facilitate the understanding of the mechanism of the descending abscesses, and will demonstrate the logic of this operative method.

According to the literature, which is fully given in my previous articles, the abscesses descending from the upper air passages and the base of the tongue, that lead to phlegmon of the neck and to affections of the mediastinum, originate mainly (1) from angina of the faucial tonsils, especially tonsillar and peritonsillar abscess, (2) following tonsillectomies, (3) following phlegmon and erysipelas of the pharynx (4) following laryngeal phlegmon, (5) following foreign bodies, (6) as secondary descending abscesses, which originate after lues, tumors, infections of the teeth, the jaw, the sinuses and other affections of the upper air passages and their adnexae.

The retropharyngeal space is formed by the interstices filled with connective tissue between the fasciae of the vertebral column and the pharynx. It continues downward within the neck, alongside of the esophagus, and is laterally connected with the parapharyngeal space, which is of great importance on account of its relation to the tonsils. The parapharyngeal space is, roughly speaking, represented by a connective tissue interstice bordered

by the pharyngeal wall, the M. pterygoideus internus, the vertebral fascia and the capsule of the parotid gland. It contains the internal carotid artery, the internal jugular vein, the hypoglossus, vagus, glossopharyngeus and accessorius nerves. The tonsil is connected through loose submucous connective tissue with the lateral pharyngeal wall (M. constrictor pharyngis and fascia pharyngea), that by itself forms the median border of the parapharyngeal space. These connective tissue spaces of the mouth and pharynx continue downward into the interstices of the neck. They are formed by the differentiation of the various fascial leaves, and contain within the loose connective tissue that fills them out, the large vessels and nerves. Of utmost importance is the middle space of the neck (spatium colli medium) located beneath the hyoid body and between the three fascial leaves. It contains the following structures: larynx, with trachea and attached to it posteriorly, and fastened by loose connective tissue to the deep fascia, the esophagus. Furthermore, it contains the thyroid gland, the common carotid artery, the internal jugular vein and the vagus nerve. The connective tissue that fills out these spaces densifies itself as a special cover around the larynx, trachea and esophagus. These connective tissue interstices of the neck lead downward to the level of the thorax aperture into the mediastinum, and upward alongside of the large vessels into the parapharyngeal space and into the retromandibular fossa.

The operative method recognized by me as a typical one is based upon the above mentioned topographic and anatomic conditions, as they decide in every case the path along which the abscess may descend. It is evident that all inflammations originating in the neighborhood of the pharynx, the tonsils, the base of the tongue and the laryngeal region, descend into the loose connective tissue interstices of the neck that surround the pharynx. *Consequently, however different the places of origin may be, all these descending abscesses act in their further course analogously, and, furthermore, may be reached by one and the same method.*

To illustrate the operative procedure, I have had made three pictures that demonstrate the different steps of the operation. The first picture shows the skin incision that reaches alongside the anterior margin of the M. sternocleidomastoideus from the level of the mandibular angle to the jugular fossa. (Fig. 1.)

The second picture illustrates the exposure of the collar mediastinum. From the lower part of the incision, through the superficial fascia of the neck, the vascular sheath is reached by

blunt dissection, and the anterior margin of the vessels is located without further dissecting the vessels. Then the *M. omohyoideus* is divided. Following the course of the vessels, the anterior collar mediastinum is exposed and sealed with a drain of iodoform gauze tampon, as is also seen in the picture. (Fig. 2.) The thyroid gland is now moved forward and towards the median line without injuring its capsule. By lifting the thyroid gland, the posterior collar mediastinum is exposed, and simultaneously the wall of the esophagus and the vertebral column appear.

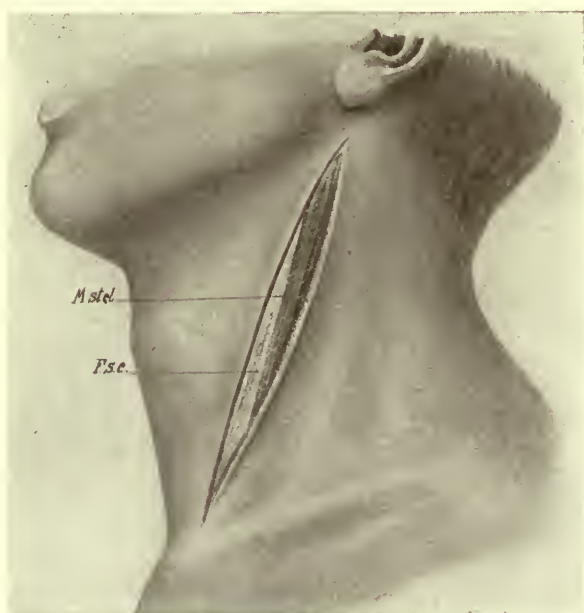


Fig. 1. M.stcl.—*M. sternocleidomastoideus*. F.sc.—*Fascia superficialis communis*.

The operative procedure up to this point is carried out both in the presence of a diseased and of a healthy mediastinum; in the first case to drain the affected mediastinum, and in the second case to seal the healthy mediastinum by iodoform gauze tampon against the possible progressive tendency of the pus (prophylactic mediastinotomy of Marschick).

The third picture demonstrates the operation proper, which can be attempted only after the sealing or draining of both collar mediastinal spaces. At the upper part of the incision, along the *M. sternocleidomastoideus*, one progresses towards the depth,

guided by the inner margin of the vascular sheath. This picture represents a certain individual instance of the typical operation, dealing with the drainage of abscesses descending from the lateral pharyngeal wall, especially the tonsils. (Fig. 3.) The lower belly of the *M. digastricus* serves as a topographic line of orientation. Within the vascular sheath, the common carotid artery and its division into the external and internal branches may be recognized. Caudally from the *M. digastricus*, the hypoglos-

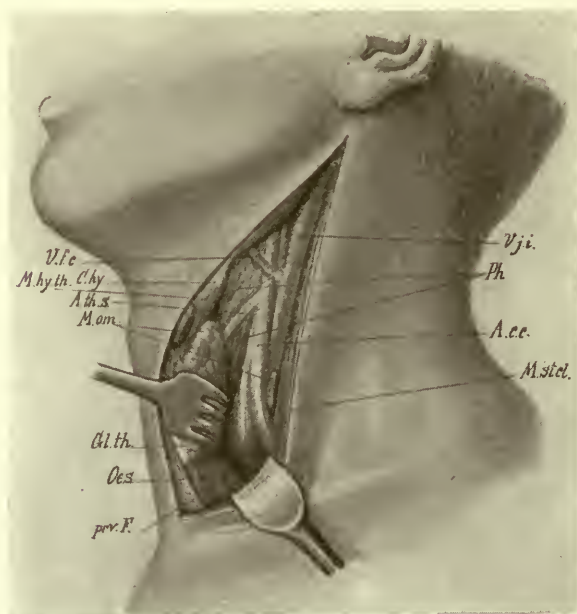


Fig. 2. V.f.c.—Vena facialis communis. M.hy.th.—*M. hyothyroid*. A.th.s.—*A. thyroid. superior*. M. om.—*M. omohyoid*. C. hy.—*Cornu hyoid*. Gl. th.—*Gland. thyroid*. Oes.—*Esophagus*. Prv. F.—*Praevertebral fascia*. V.j.i.—*Vena jugul. int.* Ph.—*Pharynx*. A.c.c.—*Art. carotid. communis*. M.st.cl.—*M. sternocleidomastoid*.

us nerve is located. In the space beneath the *M. digastricus*, as is indicated in the picture, a dressing forceps is introduced into the direction of the mandibular angle and is pushed upward into the parapharyngeal space, beneath the mucous membrane of the pharynx, and spread open in different directions (subbi-ventral method). If we have to deal with the opening and drainage of abscesses localized elsewhere within the upper air passages or at the base of the tongue, *we must, in every individual case, differ from the above described type of operative procedure, ac-*

cording to the exactly localized place of origin of the abscess. By previous diagnostic examination we determine, as well as possible, the location of the abscess, the level at which it originated and the special way the suppurative process took in the course of its progress. Due to connective tissue induration and scar formation, the origin of the descending abscess is not always easily traceable during the operation (this occurs, for instance, in case No. 5 described in my previous paper and in cases

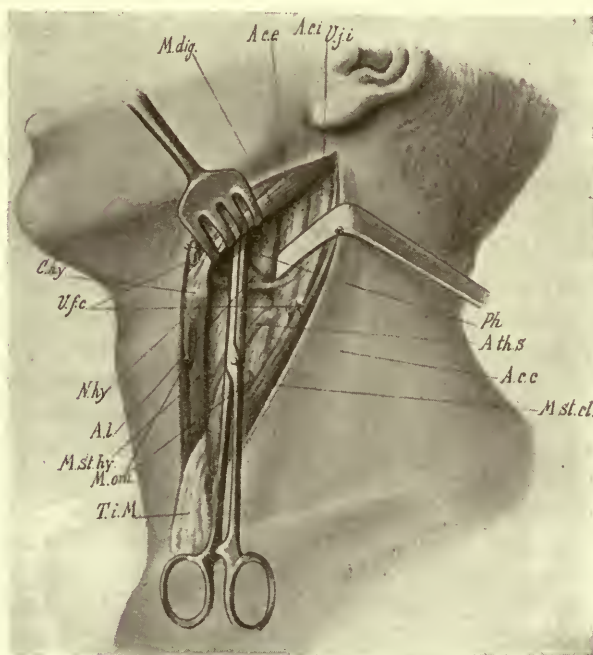


Fig. 3. C. hy.—Cornu hyoid. A. c. e.—Art. carotis ext. V. f. c.—Vena facialis communis. A. c. i.—Art. carotis int. N. hy.—N. hypoglossus. V. j. i.—Vena jugularis int. A. l.—Art. lingualis. M. st. hy.—M. strenohyoid. M. om.—M. omohyoid. T. i. M.—Tampon in mediastinum. Ph.—Pharynx. A. th. s.—A. thyroid. superior. A. c. c.—A. carotis communis. M. st. cl.—M. sterno-cleidomastoid. M. dig.—M. digastricus.

No. 1 and No. 2 of this article; the entrance to the retropharyngeal abscess was barricaded by connective tissue induration. Only by the preceding pharyngoscopic examination were we induced to remove these indurations and cut daringly through them, in order to reach the abscess cavity). Thus the originally typical method suffers an individual modification according to the special conditions present.

After the operation, the wound is left open and moist dressing

is used. Filling the wound with gauze and tamponing it should be avoided. The superficial dressing is changed daily, in order to keep the wound moist. The gauze strips within the mediastinum remain about four or five days, and are then replaced by small strips.

The operative method described above was successfully applied in the following three cases:

CASE 1. Ch. T., 11 months of age, was referred to me on February 2d, 1922, by Dr. Charles F. Fisher of Brooklyn.

Anamnesis: The child was born in the U. S. A. The parents are Russians and claim to have always been in excellent health. The child is breast fed and got its first teeth at 6 1/2 months. The present sickness is the first one the baby suffered from. About three weeks ago, the baby started to cry all night. The mother gave it castor oil and soap and water enema, without avail. The next day a physician was called, who found a temperature of 104° and rapid pulse. He also noticed a swelling on the left side of the neck. The doctor suspected peritonsillitis, and prescribed a mouth wash and internal medicine. As the child's condition grew worse, an ear specialist was called, who opened both drum membranes. An X-ray picture was taken, but proved to be negative. The child's condition grew constantly worse. A baby specialist was then consulted, who thought that an abscess was forming somewhere, but could not locate it. Meanwhile the child started to choke, especially at night, and would not take the bottle any more, and if forced to do so, would vomit immediately. Another baby specialist was consulted, who claimed that the stomach was out of order and prescribed a special diet. The baby grew weaker and weaker, the breathing became more and more obstructed. The temperature showed a daily range from around 99° to 104°. During the last few days, the child had chills. Dr. Charles F. Fisher, who saw the child in the morning, suspected a deep abscess in the neck and referred the patient to me.

Status presens: The baby is well developed, extremely pale and apparently very ill. It is gasping for air and slightly cyanotic. When given the bottle, it vomits immediately. Temp. 104°, Pulse 100.

Local pharyngoscopic examination reveals no apparent pathologic change within the throat, with the exception of a marked redness at the base of the tongue, in the region of the vallecule. Digital examination reveals a fluctuating swelling on the posterior wall of the esophagus, starting at the level of the arytenoid

cartilages and apparently reaching far downward. On the outside, there is dense infiltration noticeable on the left side, at the anterior margin of the sternocleidomastoideus muscle, starting from the mandibular angle and reaching down to the larynx. The swelling is slightly discolored and is very sensitive to pressure. There is a special sensitiveness to pressure present at the region of the jugular fossa; heart and lungs are apparently normal.

Diagnosis: Descending retroesophageal abscess with phlegmon of the neck and threatening mediastinitis. Immediate operation was advised.

Operation performed at Lane Sanatorium, Feb. 2d, 1922, 1:30 p. m., Dr. Levensgood assisting, Dr. Lane anesthesiologist. Superficial ether narcosis. The incision is made alongside the anterior margin of the sternocleidomastoideus muscle, from the mandibular angle to the jugular fossa. Ligation of the superficial vessels. Blunt dissection is made alongside the anterior margin of the muscle to the depth of the vascular sheath, whose anterior margin is located, without however opening the sheath proper. At the lower aspect of the wound, the omohyoideus muscle is severed. Following the inner margin of the vascular sheath downward, the anterior mediastinum is exposed. It is found to be normal and is sealed with iodoform gauze tampons. The thyroid gland is then pushed forward and towards the median line, without, however, injuring its capsulae. By lifting the thyroid, the posterior mediastinum is exposed. As it is found to be in a healthy condition, it is sealed with iodoform gauze tampon. Having thus protected both the anterior and posterior mediastinum (prophylactic mediastinotomy of Marschick), the abscess cavity is searched for at the upper aspect of the wound. Here too, the inner margin of the vascular sheath is located. But the tissues are very hard and indurated, and it is impossible to continue working towards the depth by means of blunt dissection.

It looked rather daring and desperate to use a knife so close to the carotid artery. But the preceding pharyngoscopic examination pointed the way. At the level of the cricoid cartilage, the indurated mass was incised right close to the anterior margin of the vascular sheath, about one millimeter from the carotid artery. The larynx was grasped with the left hand and pulled outward. When the incision had been carried about 1 cm. further towards the depth, there gushed forward, under high pressure, about 5 ounces of creamy, foul smelling pus. Starting from this point, the indurated mass of tissue was then opened

further upward and downward by means of the finger. Digital examination of the abscess cavity showed it to be about 2 1/2 cm. in width and 8 cm. in length. The vertebral column could be clearly felt beneath its fascia, but no roughness pointing to bone affection could be noticed. Two cigarette drains were introduced into the abscess cavity and the wound was left open. Immediately after the operation, the child's condition showed a remarkable change. The same evening it sat up in bed, breathing normally; it asked for the bottle of milk, which it emptied crav- ingly and without difficulty. The temperature dropped within a few hours, and the pulse improved with it. The night after the operation, the child slept quietly and restfully. The outside dress- ing was changed every day. On the fourth day the mediastinal tampons were removed, and on the fifth day the 2 cigarette drains, as there was no discharge whatsoever present. With the removal of the drain, a slight irritative cough stopped, which was due to the pressure of the drain towards the larynx. On the sixth day, the child was dismissed from the Sanitorium as cured. After one week, the wound had entirely healed by secondary intention.

CASE 2 represents a similar condition in an adult, where too the typical external operation proved to be life saving.

S. H., 30 years old, referred by Dr. Mark J. Schoenberg, April 11, 1922.

Anamnesis: Twelve days ago, the patient caught a cold and immediately noticed a swelling on the left side of the neck. This swelling, however, disappeared, but came back four days later. Since that time, the patient has felt a choking sensation, has pain on swallowing, and experiences shooting pains toward the head, back and arms. He cannot open his mouth very well and is gasping for air.

Present state: Patient has a subicteric discoloration, and is evidently very sick. There is no apparent inflammation present at the tonsils, base of the tongue, or pharynx. Laryngoscopic examination reveals slight edema of the larynx entrance. There is noticeable at the level of the *posterior laryngeal wall, on the left side, a forward bulging of the esophageal mucous membrane, which is highly inflamed.* There is severe pressure pain along- side of the anterior margin of the left sternocleidomastoideus muscle, at the level of the cricoid cartilage. On the right side, the same pain is elicited, but only on deeper pressure. The patient holds his head stiff and inclined to the left side. There is a dif- fuse infiltration in front of the left sternocleidomastoideus

muscle from the mandibular angle to the larynx, and a slight swelling in front of the right muscle. Swallowing is extremely difficult. Severe tenderness to pressure is present in the jugular fossa. The patient is slightly cyanotic.

Diagnosis: Descending retroesophageal abscess with phlegmon of the neck and threatening mediastinitis. Immediate operation is advised. The patient went home, however, and returned only late in the afternoon.

Operation at the Community Hospital, at 8 p. m., Drs. Hoidanl and Grove assisting. Anesthetist, Dr. H. Hraba. Operation began at 8 p. m. and closed at 9 p. m.

Under superficial ether anesthesia, an incision was made alongside the anterior margin of the sternocleidomastoid muscle, from the mandibular angle to the jugular fossa. By blunt dissection, aided by the use of finger at the lower margin of the wound, the anterior mediastinum was opened and sealed with iodoform gauze tampons. The thyroid gland was then pushed forward, and the posterior mediastinum exposed and sealed in the same way. At the level of the cricoid cartilage, a solid mass of indurated tissue was found in front of the vascular sheath, together with enlarged and degenerated glands. One gland of the size of a hazelnut was located at the division of the common carotid artery. First by blunt, and then by sharp dissection, it was worked towards the depth. In front of the carotid artery, without opening the vascular sheath, the retroesophageal tissue was reached by deep incision. About six ounces of foul, yellowish creamy pus were evacuated. Digital exploration showed a large cavity between the fascia of the vertebral column and the fascia of the esophagus, reaching downward about 5 centimeters and *encroaching upon the other side*. The wound was left open except for two stitches at the ends of the wound. Wet dressing was applied.

Due to the bad breathing of the patient, I was at any moment during the operation ready to perform tracheotomy. The anesthetist remarked, that the moment the pus was evacuated, the patient's color immediately changed to normal, and his breathing, which during the operation became very bad, improved instantaneously. The narcosis was a very light one, and the patient soon after the operation cried out, "Oh, I feel so much better." The next day he smoked his cigarettes and ate three hearty meals. He did not complain any more of pains. The outside dressing was changed every day and kept wet. The mediastinal gauze tampons were removed on the 4th day, and the cigarette drains on the

6th day, to be replaced by a small strip of plain gauze. On the 8th day following the operation, the entire wound, which had been left open, was closed with the exception of a small central area which closed up after one more week.

CASE 3. P. R., 9½ months old. Referred to me on April 26, 1922 by Dr. Charles Fisher, Brooklyn.

Anamnesis: One week ago the baby got sick with a cold. Two days later a swelling occurred at the right side of the neck, the next day also on the left side, but smaller than the one on the right side. The baby could not swallow at all. The temperature from the very beginning of the disease was 101°, and a few days afterwards, ranged from 99° to 105°. All this time, the baby was breathing very badly, especially at night. Due to choking, the baby would refuse the bottle, and in fact took no nourishment at all during the last few days.

Present state: Well developed baby, with very sickly expression, apparently in severe pains.

Local examination reveals inflamed tonsils and redness at the base of the tongue. Digital examination, as far down as possible, does not reveal any bulging or fluctuation. In front of the right sternocleidomastoideus muscle, there is a large swelling of doughy consistency and tender to pressure. It reaches from the mandibular angle almost to the jugular fossa, being about 10 cm. in length, 4 cm. in width and 3 cm. in height. There is a similar swelling in front of the left muscle, reaching only to the level of the larynx, and being about half in dimensions. The baby has great difficulty in breathing. Pulse 120, temperature 104°.

Diagnosis: Phlegmon of the neck due to descending abscess.

Operation performed at Community Hospital, April 27th, 2 p. m., Dr. Levingood assisting, Dr. Blumenthal, anesthetist. Incision is made alongside the anterior margin of the left sternocleidomastoideus muscle from the mastoid tip to the jugular fossa. Ligation of superficial vessels, severing of omohyoideus muscle. Opening and sealing of the anterior and posterior mediastinum in the regular way. The vascular sheath is exposed at the upper part of the wound. The tissues are all indurated and infiltrated. A big mass is found of the size of an egg (about 5 cm. in length, 3 cm. in width and 3 cm. in height), intimately connected with the jugular vein. It is dissected out and proves to be a mass of glandular tissue, containing pus and cheesy material. Right beneath where the mass has been, the jugular vein appears without a lumen. From there downward, towards the jugu-

lar fossa, the internal jugular vein is flask like dilated. In its upper part, the dilation is about 3 cm. in diameter, becoming gradually thinner after extending 4 cm. downward. The surrounding tissues are edematous. The retropharyngeal space is opened in front of the carotid. It does not contain pus, but shows edema which apparently spreads towards the other side. The parapharyngeal space is investigated by means of dressing forceps being spread beneath the digastric muscle, in the direction of the tonsil. No pus is found here.

The flask like dilated portion of the internal jugular vein is excised, from 1 cm. beneath the level of the digastric muscle to the jugular fossa. Cigarette drains are introduced beneath the digastric muscle and into the retropharyngeal space. The wound is left open, wet dressing is applied. After the operation, Murphy drip was applied, but soon discontinued, as the child took nourishment. During the next four days, the condition of the child improved markedly. On the fifth day, however, the baby showed a marked swelling of the face, with characteristic redness, temperature 104° , pulse 140. Erysipelas was diagnosed and the child dismissed from the Hospital. Within 10 days, all symptoms of erysipelas disappeared under appropriate treatment, carried out by Dr. Fisher. The widely gaping wound gradually closed by granulations, aided by adhesive plaster strips. Three weeks after the operation, the wound was closed. The swelling on the left side had disappeared 2 days after the operation. The drain in the retroesophageal space aided the absorption of this secondary edema and infiltration.

As case 4, I would like to report a case operated by myself 11 years ago and published at that time.¹ The descending abscess originated within the larynx, with a foreign body as probable etiologic factor. The external operation was performed. The characteristic feature of this method, the simultaneous prophylactic mediastinotomy, was not yet thought of at that time.

CASE 4. Mr. M. K. was referred to me on December 2d, 1911, by Dr. S. Tandlich.

Anamnesis: The patient had been perfectly well until five days ago, when suddenly he became aware of pain in the throat, which gradually became more excruciating in character. Attempts at swallowing water or his saliva caused agonizing pain in the throat, which extended up into his right ear. The pain was felt distinctly on the right side. A history neither of syphilis nor of tuberculosis was obtainable; no history of injury or foreign body in the throat.

Status Praesens: Middle aged man, not very robust, apparently suffering acutely. Externally, no swelling was visible. The right submaxillary region was tender on pressure. The ear drums were normal. Upon depressing the tongue, a swelling was seen occupying the space between the base of the tongue and the epiglottis. It was of a deep red color, had a lobulated surface, was hard to touch, and did not fluctuate. It was of the size of a walnut. The anterior and upper surface of the epiglottis formed part of the growth, and was displaced backward by the latter, so as to completely occlude the entrance to the larynx. The pharyngeal mucous membrane was reddened on the right side, but there was no bulging. Temperature, 101° F., pulse, 97.

Tentative Diagnosis: Acute perichondritis of the epiglottis.

As there was no ulcer present on the epiglottis itself (this is given as the most frequent cause of such conditions), I concluded that there must be some lesion in the larynx proper. The growth was incised, but no fluid was evacuated.

Treatment: Hot antiseptic gargle, and hot flaxseed poultice externally.

December 3d, the subjective symptoms were unchanged. Upon inspection, it appeared that the swelling had subsided a little and thus permitted a view of the larynx. A cyst like swelling of whitish color, the size of a hazel nut, was now seen projecting into the right side of the larynx, having its base on Santorini's cartilage. There was a diffuse infiltration of the right arytenoid cartilage and the posterior part of the cricoid cartilage, extending forward towards the annular portion of the latter.

Suspecting pus, the writer made several exploratory incisions with a protected laryngeal knife, but none was detected.

The following day, a swelling the size of a fist appeared just below the jaw, the pus evidently having invaded the cellular tissues of the neck. No fluctuation, however, was discernible.

Operation: On December 7th, at St. Mark's Hospital, under general anesthesia, an incision was made over the swelling in front of the sternomastoid muscle and at the level of the hyoid bone. By means of blunt dissection, and with the aid of the index finger, the tissues were separated until a large abscess cavity was reached and emptied of a considerable quantity of yellowish, foul pus. Within the cavity, the carotid artery could be felt pulsating. On the inner side, a roughened area of cartilage proved to be the partly destroyed annular portion of the cricoid cartilage. While examining this area, the finger slipped through the wall of the abscess and a communication of its cavity and the interior of the larynx resulted. A cigarette drain

was inserted and made to protrude slightly into the larynx, thus effecting through-and-through drainage. The patient improved rapidly. The drain was shortened daily until its final removal one week afterwards.

Etiology: As regards the possible etiology of the abscess, the pus of which contained streptococci in chains, both syphilis and tuberculosis may be excluded from consideration. The Wassermann reaction and also the von Pirquet test gave negative results. The one point which may aid in clearing up the obscure etiology is the fact stated rather late by the patient, that when he had been sick a few days he had spit up a hard mass (foreign body?).

It is likely that this foreign body had been the direct cause of the abscess formation. Thus an infection near the right arytenoid cartilage caused a perichondritis of the same and of the annular portion of the cricoid cartilage. From here the infection spread to the cartilage of Santorini, and thence to the anterior part of the epiglottis and to the deep tissues of the neck.

EPICRISIS.

The first two cases are almost identical. The 11 months old baby showed a discolored painful infiltration on the left side of the neck and a fluctuating swelling on the posterior wall of the esophagus. In the 30 year old man, there was a similar infiltration on the neck, but on both sides. The forward bulging of the esophageal mucous membrane pointed in both cases to a descending retroesophageal abscess. The level of this esophageal swelling, as mapped out by pharyngolaryngoscopic examination, pointed the way for the location of the pus. Thus only could one safely cut through the mass of indurated and infiltrated phlegmonous tissue surrounding the vascular sheath and separating the latter from the abscess cavity. The typical operation saved the life of these two patients, who were critically ill. The prophylactic mediastinotomy prevented the threatening complications from the mediastinum and the vital organs.

In the third case, a 9 1/2 months old baby, the swelling of the neck was due to phlegmon of a big mass of glandular and connective tissue and to the consequent flask like dilatation of a large portion of the internal jugular vein. Only through the typical method could the flask like abnormality of the jugular vein (a pendant to which I had only found in a case where the sinus transversus and jugular bulb has been blocked off by a thrombus) and the pathologic mass of tissue be removed from the vascular sheath. Prophylactic mediastinotomy prevented threat-

ening complications. In this case the descending abscess started from the inflamed tonsils, most likely from an infection of the vallecula at the base of the tongue, which in children appears to be a frequent, though mostly overlooked, focus of infection. The descending abscess in case 3 was, also, caused by a "cold".

The etiology in case 4 was apparently a foreign body. Such a case is especially dangerous, as here the phlegmonous infection tends to spread rapidly. The timely external operation saved the patient's life, as he was in danger of suffocation, due to the abscess cavity encroaching upon the larynx.

To complete the record of the cases of descending abscesses operated upon by the typical operation, I will add an abstract of the epicrisis of the 8 cases fully described in my previous papers. From a total of 12 cases operated upon, 9 recovered. In three cases, the patients were brought to the clinic in such a precarious condition, that in one case no attempt at an operation could be made, and in the other two no surgical interference could be of help. However, the postmortem examination performed in these three cases illuminates the etiology and mechanism of these descending abscesses, and impresses the necessity of timely performing the typical external operation, combined with prophylactic mediastinotomy.

EPICRISIS OF THE PREVIOUSLY REPORTED CASES.

In CASE 1, we have to deal with a diffuse retrotonsillar phlegmon that has been twice incised without success. One day after admittance, there develops a swelling on the neck, reaching from the mandibular line to the larynx, with rise in temperature and increased painfulness of the swelling. During the operation, the mediastinum is sealed and the focus of suppuration is opened by pushing the dressing forceps beneath the belly of the *M. digastricus*, towards the pharyngeal mucous membrane. The sealing of the mediastinum was done just in time, as the perivascular tissue was already infiltrated and edematous.

CASE 2 represents a deep phlegmon of the neck, consequent to an abscess of the base of the tongue, that was induced by lacunar tonsillitis. The indication for mediastinotomy and for the typical opening alongside the vascular sheath was given by a marked edema of the pyriform sinus, the rapid pulse and the painful swelling of the neck. The vascular sheath proved to be already closed by induration, and there was found an abscess cavity reaching to the pyriform sinus, the larynx and the vallecula.

The first two cases prove that absolute recovery is possible when the surgical operation is performed in due time.

CASE 3 shows the consequence of not performing a timely operation. Especially favorable circumstances of very rare occurrence prevented, however, a fatal termination. A woman of thirty years of age, for twelve days noticed a rapid enlargement and an inflammation of a swelling of the neck, that remained when she left the hospital, five weeks ago. Her condition, when admitted for the first time to the hospital, was as follows: The neck at its anterior part, alongside the M. sternocleidomastoideus down to the jugulum, showed a marked infiltrate that was hard, painful and reddish blue. At the left side, there was a pronounced swelling and infiltration of the lateral pharyngeal wall that reached down to the pyriform recess. The lingual side of the epiglottis was also edematous. The strictest indication was, therefore, given for the typical opening of the vascular sheath, with drainage of the abscess and sealing of the mediastinum. Before, however, the intended operation was performed, the abscess broke open through the mouth. All symptoms disappeared, but a swelling at the left side of the neck, of the size of a child's fist, remained. Five weeks later, when again admitted to the hospital, this swelling suddenly had reached the size of a coconut, becoming lividly discolored and fluctuating. Simultaneously, there was present a swelling of the left tonsil and the adjoining pharyngeal wall. When the fluctuating swelling of the neck was opened externally, there appeared a large abscess cavity that was apparently walled off from all sides. Further operation, therefore, seemed unnecessary. The walling off of the abscess cavity saved the patient from sepsis. To wait for such a favorable occurrence is, however, too great a chance, that in most instances may endanger the life of the patient. At her first stay at the hospital, the patient should have had performed the external surgical operation that later on, through acute acerbation, endangered the patient's life; thereby absolute recovery would have been brought about without the infiltrate of the neck remaining.

IN CASE 4, we have to deal with a set of false teeth that had been swallowed, and became fixated within the hypopharynx. The most characteristic external symptom was a very large emphysema of the skin, that was red and sensitive to pressure. Through the typical operation, there was found on the exposed pharynx a necrotic, bad smelling area of the size of a child's hand, combined with phlegmon of the vascular sheath and edema in both mediastina. These life endangering complications caused by a foreign body give the strictest indication for the typical operative method, which should be resorted to even when only slight external neck symptoms occur, following the swallowing

of foreign bodies. The necessity of performing a tracheotomy during the operation impresses one deeply with the danger of neck complications caused by foreign bodies. The recovery was quick and is doubtless due to the timely performance of the typical operation, whereby the necrotic and phlegmonous area was drained, and the already edematous mediastinum was sealed.

A foreign body, too, is the underlying condition in CASE 5. It could, however, not be located, either through local inspection or through the Roentgen picture. There were evident the most severe general symptoms and marked local manifestations (diffused swelling of the posterior pharyngeal wall and of the carotic triangle at the level of the upper margin of the thyroid cartilage). It is important to note that laryngoscopic examination had revealed the oozing of greenish-yellowish pus from the left pyriform sinus at the level of the arytenoid cartilage. This finding proved to be of great help in guiding the way during the typical operation. Within the indurated and edematous tissue, no pus could be found. The above laryngologic finding, however, induced the progress towards the lower side of the hypopharynx near the vertebral column. And here an abscess cavity filled with blood coagulum and crumbs of very bad smelling pus was found, in which, transversely in front of the vertebral column, there was imbedded a bent and entirely rusted sewing needle. The original incision had to be enlarged downward into the healthy tissue, due to pressure sensitiveness in the anterior mediastinum. There occurred complications of the lung, right sided paresis of the sympathetic nerve and atrophy of the right sided shoulder muscles, which were relieved by corresponding treatment. The symptoms disappeared within a short time, and the patient recovered entirely. This case points to the importance of a thorough laryngoscopic examination and of its interpretation during the operation.

The following three cases, all terminating fatally, show how rapidly the pathologic process may spread from the mouth, pharynx and larynx to the mediastinum, and from there to the vital organs.

Only a very early operation would have saved the life in these cases. When the patients came to the Clinic, the deep complications of the neck and chest, and the sepsis already present, gave no chance to save their lives by a typical operation, even with drainage of the mediastinum.

CASE 6 is that of a sixty-year old man, who was admitted after the occurrence of the first symptoms (swelling, difficulties in

swallowing). Laryngoscopic examination showed no important pathologic changes within the throat and larynx. Notwithstanding this fact, there is revealed by the postmortem examination suppurative left sided tonsillitis, with an abscess of the size of a bean. One of the abscesses, located at the base of the tongue, ruptured towards the vascular sheath and spread its pus not only along the same, but also towards the mediastinum, pleura and lung. Parenchymatous degeneration of the organs and tumor of the spleen was there present as in all cases that came to autopsy.

IN CASE 7, an eight months old baby, the pharyngeal abscess *originated from the vallecula*. The inflammatory swelling and suppurative decay of the parapharyngeal, submaxillary and paratracheal glands show the pathway the pus took in order to cause suppurative mediastinitis, anterior and posterior, pleuritis and pericarditis. This case shows clearly the necessity of performing operation immediately, whenever, in babies, the first symptoms of phlegmon of the neck occur. No operation was performed.

IN CASE 8 we have to deal with a forty year old man. The abscess evacuated through the mouth. Notwithstanding this, the inflammatory edema of the hypopharynx and the phlegmonous swelling of the neck did not disappear. During the immediately performed operation, tracheotomy had to be resorted to, on account of the patient's asphyxia. The discoloration of the pus that was evacuated by the subbiventral method, the gelatinous, fibrinous exudate of the deeper portions of the neck, the severe general symptoms and the rapidly appearing dullness over the mediastinum and the apices, excluded from the beginning a favorable result of the operation. The autopsy finding revealed as primary focus numerous tonsillar abscesses filled with discolored pus, with very marked edema of the glottis. In a very distinctive way, the path of the pus can be followed on the one side from the tonsillar abscesses into the mediastinum and into the pleura. On the other side, it bores alongside the prevertebral fascia down to the promonotory and alongside the periesophageal connective tissue down to the cardia. In this case, as in many similar ones, we have apparently to deal with chronic tonsillar abscesses that by acute exacerbation lead, in a short time (in this case within two days), to edema of the glottis and to the descending of pus with consequent sepsis.

These autopsy findings show how rapidly the abscesses descend into the neck and mediastinum; they impressively teach us not not to endanger the patient's life by protracted conservative treatment.

The typical operation, reaching the original suppurative focus alongside the course of the vascular sheath, is a logical procedure, because it corresponds to the topographic anatomic conditions. It should always be combined with opening of the mediastinum (collar medianstintomy), followed by sealing or drainage of the same, depending upon whether it is in a healthy or diseased condition. At the early stage, conservative treatment may be tried, such as local antiphlogistic therapy, incision within the upper air passages or, at times, also external incision without opening of the mediastinum. The patient must, however, be most closely observed, and one must be ready to immediately perform the typical operation in case of emergency. Such cases were reported in my previous paper.

The direction of the progress into the depth, from the typical opening alongside of the vascular sheath, depends upon the localization of the original suppurative focus, whereby the most exact laryngoscopic examination is of utmost importance.

If the topographic anatomy of the region is mastered, the technic is simple.

Acting under correct indication, and if the operation is performed in time, the result is always favorable.

This typical external method for the relief of abscesses descending from the upper air passages and the base of the tongue should be preferably performed by the well trained laryngologist, who takes special interest in this kind of work, for he alone is thoroughly orientated by the all important pharyngolaryngoscopic examination, which increases the value of his knowledge of the topographic anatomic conditions of these regions. Besides, the modern rhinolaryngologist ought to master the entire surgery of his special field, including all major operations.

Since I read my first paper on the subject, before the Section on Laryngology, of the New York Academy of Medicine, and especially since its publication in the Laryngoscope; I received numerous communications, written and verbal, regarding the fatality of these descending abscesses, when treated by the old method. Phlegmon of the neck, with mediastinitis and fatal sepsis, due to hitherto overlooked or disregarded descending abscesses, were reported to me by dentists (extraction of teeth and dental infections), otologists (mastoiditis), rhinologists (tonsillitis, peritonsillar abscess, sinus affections, foreign bodies, etc.) and general practitioners (lues, tumors, etc.). It is my hope, that through my endeavors, a stimulus has been given to the profession for a correct and early diagnosis of this dangerous condition. An apparently simple case should not be trifled with,

and a desperate one should be given a chance. This method is submitted to the surgeon, especially to the well trained rhinologist, for a fair trial to pass upon it in cool judgment. Thus a measure conceived and advocated in enthusiasm will gradually find its right place in our armamentarium toward the relief of human suffering.

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DISCUSSION.

DR. EMIL MAYER, New York, N. Y.: I have very little to add. This subject has been brought up by Dr. Glogau before, and we have read a great deal of it in the journals. He is naturally enthusiastic about a condition which confronts us sometimes, and is undoubtedly a very grave condition. Postoperative conditions are sometimes seen with an enormous phlegmon of the neck, and when it comes to the question of where this was originated, I would not wish to go on record as saying that it originated in Dr. Hajek's Clinic, for I am sure our American surgeons have shut off the mediastinum, and I think his claim is that the shutting off of the mediastinum is what shuts off the infection.

We are indebted to Dr. Glogau for showing that in desperate cases that need desperate means, the external incision is the only remedy when you suspect the formation of the abscess, where the patient's condition is so feeble that he cannot stand anything like an internal incision, or where the amount of brawny infiltration is so great that you must have an external drain.

DR. JOHN F. BARNHILL, Indianapolis, Indiana: I wish to make a few remarks regarding diagnosis and treatment. Diagnosis here is as important as anywhere in surgery, I believe. In cases such as described, where patients have a rise in temperature, even though there is not much complaint other than stiff neck, the symptoms demand the most thorough investigation we can give. The first symptom is quite often a stiff neck. It appears before one may be able to detect any tumefaction. When one remembers that the accumulated pus lies deeply in the neck, is covered by muscle and layers of dense fascia, it becomes easy to understand that exact diagnosis becomes difficult, as it is often impossible to make out either fluctuation or tumefaction in the early stages. Hence we should make use of the very early symptoms of stiff neck and rise in temperature.

Incision of this abscess through the mouth or upper pharynx in some cases is successful. When the burrowing has made a bag beyond our ability to see and enter, then in all cases there should be an external incision, for in all cases it is the safest thing to do, because then you can do the work Dr. Glogau has described, and will almost without exception cure the disease.

I do not know where the method Dr. Glogau described originated. No doubt many knew and practiced it before the Vienna clinic described it. I should like to have Dr. Glogau explain the exact method he employs for closing off the mediastinum.

DR. JOSEPH C. BECK, Chicago, Illinois: Of course everyone recognizes the great importance of this condition and has seen the cases. I have talked with Dr. Glogau, and think the question of closing off the mediastinum is one of the most important things. He says seal off the mediastinum. Now nature does that, and his attempt to improve on that by breaking down barriers is something that most surgeons are getting away from, so I do not consider his attempt to make a two step operation to prevent infection getting into the neck a good point. I have seen many cases, where if they did reach the abscess, they make the drain upward instead of making a long incision and making the drain downward.

The question of differential diagnosis is very important, but time will not permit my taking it into consideration.

I wish to make the point, that the operation of sealing off the mediastinum, or laryngectomy, is not being done, and the breaking downward with the finger toward the mediastinum I think is wrong.

DR. HARRIS P. MOSHER, Boston, Mass.: Deep cervical abscess is not a new subject, in spite of the enthusiasm injected by the speaker. Most of the cases are easily recognized and easily dealt with. I agree with what Dr. Beck said about the adhesions produced by Nature, and doubt whether they should be broken up.

There is another type of case which is not so easily recognized. I thought I had discovered it, but found I had not. This is the type of case, where some infection has been present in the throat which has quieted down, only to be followed by a rise in temperature, which indicates a very septic case. Patients have died because they have been classed as general sepsis and allowed to go at that. These are really cases of deep internal abscess surrounding the internal jugular vein. After a throat infection, if you have a septic temperature, you should cut down and examine the vein and act according to the condition found.

DR. OTTO GLOGAU, New York, N. Y., (closing): I do not claim that opening from the outside or shutting off the mediastinum is new. I only claim that the general use of this typical method will make it universally valuable in reaching these descending abscesses. I was told by Dr. Beck, that in America, Dr. Crile closed up the mediastinum first. I was under the impression that Dr. Marshick did it first.

The point brought out, that the stiff neck is an early symptom, is a good one. Whenever we have this stiff neck following infection of the throat, we must be on the lookout.

The breaking down and sealing of the mediastinum is done by introducing a piece of gauze into the anterior and posterior mediastinum. If there is any pus or infiltration a drain is introduced deeper downward. If there are no such signs, a piece of gauze is used to close it up. We frequently find the condition of the throat healed up entirely, and suddenly the patient gets symptoms of sepsis, or we may not find in the history anything pointing to a previous sore throat. It is not always necessary to make a prophylactic mediastinotomy. In my first paper, I published a number of cases treated conservatively; but in all serious cases, where the patients have formerly died, this operation should be resorted to. Every effort should be made to find the source of the pus and drain it.

SINUITIS FROM SWIMMING.

RALPH A. FENTON, A.B., M.D.

PORTLAND, OREGON.

With the very great increase of public interest in swimming, diving, and the use of public tanks and bathing places, it is often alleged that disease is spread by such resorts. Spasmodic efforts to regulate public baths have been succeeded by a rather coherent policy of water examination by health authorities, and by insistence upon preliminary showers, provision of heat sterilized suits, as well as chlorination, ultraviolet ray treatment, and other bactericidal measures for the water itself.

Nevertheless, the incidence of ear, nose and throat disease following swimming is still commonly noted by rhinologists, and is often blamed upon dirty water. Certain fallacies in this generalization have caused us to examine statistical findings in our own city and in other fields, and to present a series of typical cases, so that free discussion of the subject might occur before this Academy.

BACTERIAL CONTENT OF CITY AND TANK WATER, PORTLAND, OREGON

Date	Location	Bacteria	Sewage
7/11/22	Council Crest reservoir.....	Sterile	none
6/ 7/22	Kings Heights reservoir	Sterile	none
6/12/22	Y. W. C. A. tank.....	30	none
6/29/22	M. A. A. C. tank.....	1560	none
6/29/22	Kts. of Columbus tank	4800	none
6/29/22	Broadway Natatorium (public)....	2880	none
6/29/22	McMahon tank (school)	720	none
6/29/22	B'Nai Brith tank	3600	none
6/29/22	Shattuck tank (school)	4800	none
6/29/22	Couch tank (school)	3600	none
6/29/22	Willamette River at Sellwood Ferry (above sewers)	Sterile	none
6/29/22	Willamette River, opposite Brooklyn sewer	300	none
6/29/22	Roadside ditch, Bertha Station (Typical stagnant pond)	300	none

The bacterial content of public tanks in Portland runs from 30 to 4800 per cc.¹; that of the Willamette River as it enters the city limits, after traversing a populous valley for 135 miles is, in June, 300 per cc.; the river water off shore from the mouth of one of the city's largest sewers contains only 2000 per cc.²

Published figures for the River Thames are from 277 (April) to 2075 (January); for the Illinois River at Ottawa 6300 to 8200 (May); and for the Mississippi at New Orleans 805 (August) to 3597 (April)³. The initial purity of Portland's drinking water, which plates sterile throughout the year, and is brought thirty-five miles from a closed forest reserve near Mount Hood, doubtless is responsible for the low tank figures. No sewage infection was found in tanks or river. It is of course relatively hard to grow for differentiation of the bacteria toxic to human beings, from the ordinary counting plates; for accurate identification, animal experimentation is necessary, and not much work has been done in this field. Overgrowth of those germs which are viable in cool water for long periods, not requiring warmth or serum media for favorable growth, is responsible for much of the negative information regarding human disease germs in water. But, by the same token, it is scarcely possible to conceive that human germs of highly toxic action upon the respiratory mucosa can multiply under conditions prevailing in swimming tanks, rivers, or lakes. Low temperatures, unfavorable media, antiseptic agents, and the action of air and sunlight upon agitated masses of water, assure the subdivision and disinfection of most of these contaminations. Samples from various depths count very much the same.

J. W. Robinson⁴ mentions certain effective rules of the California State Board of Health, requiring that proper and convenient places for expectoration by bathers be provided; that surface contamination shall be skimmed off; that the water shall flow off at various levels continuously; and that all persons known or suspected to have infectious diseases shall be excluded. He asks pertinently who will do the excluding, and, in common with Walker⁵, suggests enactment of law making it a misdemeanor for an infected person to enter a tank.

A committee of the American Public Health Association has recently reported⁶ upon a questionnaire regarding bathing places, which has been sent to 2,000 eye, ear, nose and throat specialists.

AMERICAN PUBLIC HEALTH ASSN. QUESTIONNAIRE—ANSWERS
BY 600 OF THE 2000 EYE AND EAR-NOSE-THROAT
MEN ADDRESSED

	No. cases reported:
Miscellaneous nasal infections	31
Colds	223
Coryza	6
Rhinitis	25
Sinus infections	72
Ethmoiditis	1
Epidemic of sinuitis	1
Meningitis (fatal)	4

Tanks were considered important means of spreading "colds" by 67 per cent. of the 600 men who made detailed answers. Sinus infections were reported by 72; one man reported an epidemic of sinuitis. 145 cases of otitis media were reported; 4 cases of meningitis were attributed to nose or ear complications. (It is of course likely that many cases reported as simple "colds" were really acute sinus or ethmoid infections). The whole difficult matter of protection of tank water and of testing for the germs of upper respiratory and conjunctival disease was referred to the Laboratory Section of the Am. Public Health Assn., for report at this year's meeting.

From our own cases we have selected four rather typical examples of infections following swimming, which will be reported briefly:

CASE 1. *Acute left ethmoiditis and maxillary sinuitis; latent chronic left frontal sinuitis; frontal lobe abscess; bilateral jugular thrombosis. Fatal:* N. L., white boy, 19, referred by Dr. R. B. Stephenson of Centralia, Wn., who had opened his left ethmoid, removed part of the left middle turbinate, and washed out the left antrum. This had been done because of severe acute sinuitis with frontal headache, following upon several months of swimming and high diving, mostly in rivers and stagnant ponds, never in a public tank. Yellow pus flowed freely after this drainage, from the left frontal, with temporary remission of symptoms. In a few days, however, temperatures rose to 104° and upper lid edema pointed to orbital extension, and Dr. Stephenson brought him to Portland. Frontoorbital radiographs were inconclusive.

An extensive Luc-Ogston external frontal operation was done at once, Oct. 13, 1920. Free nasal drainage was found, and it was assumed that a tendency to point toward the

external angular process through the upper lid would cease, following the open nasal channel. Temperature went down to 99° for a day or two, but speedily rose to 104° and over, with a relatively slow pulse (Fig. 1). The optic discs were somewhat choked, especially in the left eye, so that after negative lumbar puncture, diagnosis of frontal lobe abscess was made. Cord like swelling and sternomastoid soreness, with a white count mounting to 17,500, 87% polymorphonuclear, caused the suspicion of right jugular thrombosis.

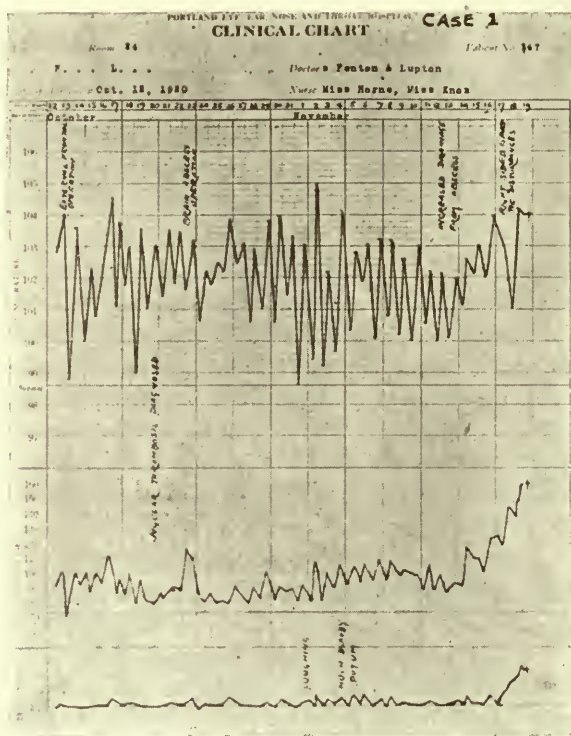


Fig. 1.

The entire inner wall of the left frontal sinus was removed Oct. 23, 1920 by Dr. Dillehunt (Fig. 2), opening a large epidural abscess, which gushed out under pressure, accounting for the constant flow of pus into the nose through dehiscences not found at the first operation, and also for the fistulization toward the outer side of the orbit.

Metastatic lung involvement was expected; the jugular soreness became bilateral. Fibrinous pleuritis and broncho-

pneumonia became manifest, especially on the right side. Remarkable stimulation was produced by several intramuscular injections of whole blood, by Dr. Bean. The patient's mental condition remained bright at all times. Some herniation of the frontal lobe took place, partially collapsing the half-centimeter soft rubber drain, through which dichloramin oil had been injected daily, with almost complete cessation of the yellow discharge. A stiffer tube was used, but about three weeks after the brain abscess was drained, heavy grumous bloody material escaped one morning, and it was feared that this meant an extension of infective thrombosis beyond the abscess area. Next day, the right arm was paralyzed, and aphasic phenomena and semicoma a day later mapped out the march

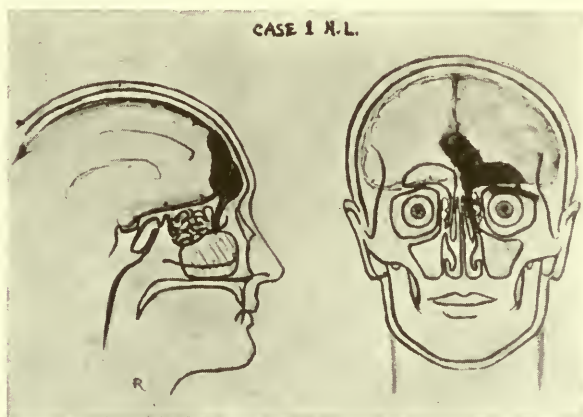


Fig. 2.

of meningeal invasion. Death came on the 27th postoperative day.

Postmortem by Dr. Menne the same evening disclosed an abscess extending vertically about eight cm. from the left orbital roof, and diagonally to the right across the longitudinal sinus, eroding the inner table of the frontal bone about two mm. The entire left hemisphere was compressed by a quantity of pus filling the dural cavity. Pus had burrowed through the orbital roof, travelled subperiosteally toward the outer angle, to appear under the eyebrow. There was no pus in the ethmoids, maxillary antra, sphenoids, or right frontal.

Most unusual was the finding of complete and organized right lateral, sigmoid and jugular thrombosis; and incomplete organized thrombosis of the left sigmoid and jugular.

There were hundreds of lung abscesses; also bilateral, coalescing bronchopneumonia, marked acute hemorrhagic and fibrinous pleurisy, acute tracheobronchial lymphadenitis, and marked cloudy swelling of the parenchymatous organs. The chief organism involved was a hemolytic streptococcus.

CASE 2. Acute left ethmoiditis; orbital cellulitis; coma; radical external ethmoid operation; recovery: A. K., white boy, 11, poorly nourished, sent in to Multnomah Hospital, Sept. 16, 1920, on account of pain and swelling over left eye following swimming in river about a week previous. Lids were swollen on day prior to admission. Temperature was 99.8°, leucocytes 11,000. Acute anterior ethmoiditis was diagnosed, and as it did not improve with saline hot nasal irrigations, and seemed to be pointing toward the inner end of the left upper lid, this

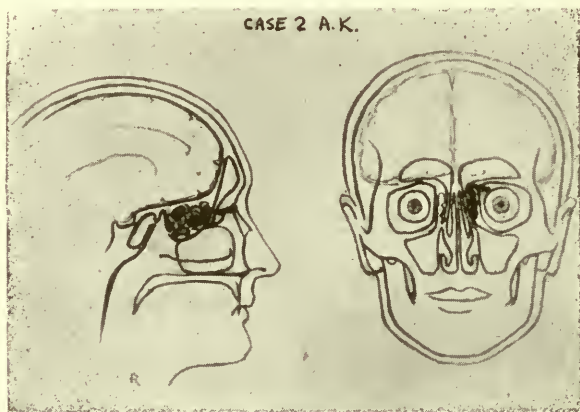


Fig. 3.

region was incised freely, Sept 29, with discharge of much staphylococcic pus (Fig. 3). The fistula came from the anterior ethmoid, and washed readily down into the nose. Under dichloramin oil irrigation, this fistula ceased to discharge pus after two weeks, and as the temperature remained normal, he was taken home Nov. 1.

Thirty-five days after the first incision, on Nov. 5, he was brought back, very dull mentally, temperature 99°, white cells 13,000, pulse 68. The ethmoid still showed dark in the radiograph. As the temperature rose slightly, the pulse reached 62 and 64. Radical external ethmoid was done on the left side, Nov. 8, opening posterior cells freely; there was little pus in front, but much in the posterior group.

The highest postoperative temperature was 100.8° , next day; the semicoma disappeared at once. Diplopia persisted for ten days. This boy left the hospital well two weeks after the radical operation, and is still well; he has quit swimming.

CASE 3. *Acute left ethmoiditis; orbital cellulitis; double frontal sinuitis; external radical frontal and ethmoid operation; exposure of dura; recovery.* A. H., white boy, 12, robust, admitted to Multnomah Hospital December 12, 1921. This lad had suffered from pains in his face and over his eyes following diving during the preceding summer. He had been sick for two months, and a swelling over the left eye had been opened by a neighbor, four months before admission. Yellow strep-

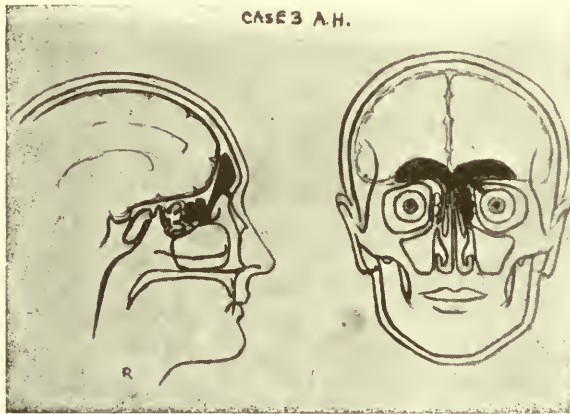


Fig. 4.

tococcic pus had discharged from the fistula since. Temperature was 97.8° , pulse 74, white count 9500; radiograph showed dark left ethmoid, with no apparent frontal involvement.

Radical external left ethmoid operation Dec. 15, 1921 by Dr. Lupton (Fig. 4); there was little posterior involvement, but a rather large left frontal, placed almost horizontally, with no extension upward into the frontal bone, was found to communicate directly with the right frontal. The partition between was loose, coming away as a sequestrum. An area of dura 1 by 2 cm. was exposed above the left frontal.

Postoperative temperature ran from 97° to normal. There was no diplopia, and the boy left the hospital nine days postoperative, and has been well since his discharge from office care a few weeks later.

CASE 4. *Chronic right maxillary sinuitis; chronic bilateral ethmoiditis; chronic catarrhal otitis media. Acute exacerbations;*

radical antrum and internal ethmoid operations; recovery. M. W., white woman, 32, an expert swimmer and exhibition diver for some years, had been troubled with head discharges for several months prior to the autumn of 1919; hearing grew worse after each season of diving. She liked to dive because it "cleared out her head," but she was always careful to expectorate mucus into the side gutters of the tanks. Her frontals were clear, antra and ethmoids dark (Fig. 5). Free, foul, curdy pus was irrigated out of the right maxillary, which yielded to treatment only to recur promptly whenever she began diving.

Radical right antrum (Caldwell-Luc) was done January 17, 1922, with removal of much polypoid mucosa and prompt

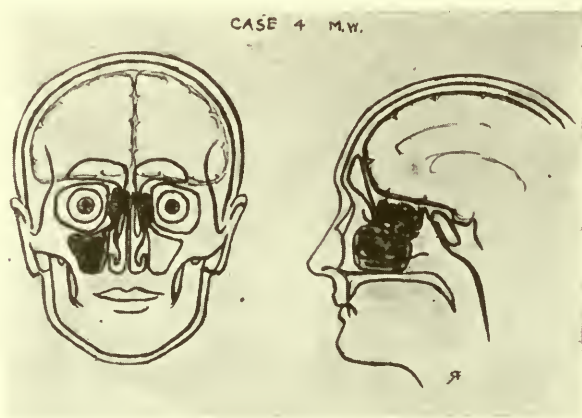


Fig. 5.

recovery. The streptococcus predominated in this infection. In spite of frequent warnings, she swam a few times more, causing such ethmoid exacerbation, that double Mosher ethmoidectomies were done in June 1922.

Practical cessation of pus has already taken place; the patient has already gained fifteen pounds, and hears about ten percent better than before the radical antrum. She does not swim now.

Such cases have abounded in the literature, but have infrequently been ascribed so clearly to swimming and diving. Cobb⁸, in 1908, stressing the fact that those who take tub baths do not put their heads under the water, cites a boy of 15 who developed ethmoiditis after five visits to a public tank, a man of 20 with acute otitis media, and a boy of 14 with double eth-

moiditis and double suppurative otitis, having tender mastoids. All recovered and remained well when they stopped swimming.

Wilkinson⁹, in 1912, and Hope¹⁰, in 1914, cited a number of cases before the Royal Society of Medicine. Wilkinson reports a man of 19, with frontal sinusitis existing for two years following diving; a woman of 32, with a suppurating maxillary antrum for six months following a "ducking" in a public tank; a boy of 17 with chronic ethmoiditis who returned from the seashore with an "orbital abscess"—osteomyelitis of the frontal bone, both frontals filled with pus, bilateral subdural abscesses, left frontal lobe abscess, and death two months later; and a boy of 15, delirious, with "orbital abscess"—double frontal; "pyemia of the lungs;" following one day after swimming in baths, and fatal one week later. The discussion turned upon strong prohibitory measures against permitting children with nasal discharges to enter public bathing places, even at the seashore.

Hope reports two frontal cases; a man of 20, swimming four days before, orbital pain and swelling next day, found to have a maxillary antrum and an orbital abscess with fistula down from the frontal floor. Discharge recurred until a radical Killian was done, when the dura was found exposed, two months later; well after three months. His second case was a girl of 14, of similar history. Polypoid granulations pointing to chronic involvement were found on her dura where exposed. This case had no maxillary involvement. It was developed that both these cases dove feet first, and was again maintained that no person with an existing nasal suppuration, however mild, should swim in a tank or pool.

The normal protective mechanism of the sinuses, as stated by Skillern¹¹, includes (1) the ciliated epithelium, with its wave like action toward the ostia; and (2) the inhibitory action of sinus secretion upon germ growth.

Swimmers and divers introduce the following factors which interfere with this protective apparatus:

(1) Destructive action of plain water upon cells; by osmosis, drawing out saline elements, causing edema and acidosis of cells, loss of ciliary activity and eventual cell death. This "irritative action of water" causes lowered local resistance. The specific protective substance of the sinus secretion is diminished and eventually lost, by being forcibly washed away, and later inhibited by the edema and chemical degeneration of the secretory cells.

(2) Washing into the sinus of pathogenic and saprophytic germs from the more highly immunized direct airways of the patient's own nose and throat; retention of these pathogenic germs in a warm cavity lined and often closed off by edematous cells with impaired resistance. Infestation of many persons by streptococci with easily increasing virulence is frequent since the "flu" epidemic of 1918. Germs of vegetable decay found mainly in water, multiplying by preference at low temperatures and in the open air, must not be considered to have much to do with sinusitis. Nor is it wise to assume that germs from other persons are directly to blame in all cases. Too many people swim alone, or in large bodies of outdoor water, and are nevertheless infected. Contamination from other swimmers directly is made less likely by the coolness of the water, its constant agitation, and the consequent high dilution of sputum found in it. The assumption that most persons succumbing to sinusitis after swimming are poisoned by their own nasopharyngeal germs, mechanically driven deep into these cavities under conditions favoring rapid growth and retention of secretions, has much justification.

(3) Direct trauma to ostia, to mucosal linings, and to bony dehiscences by forcible inrush of water, especially if chlorinated, and by the forcible outrush of air bubbles. Increase of pressure in deep diving, with the bubbling and squeaking and persistent headache reported by certain swimmers, must also be reckoned with.

Prevention of sinusitis from swimming would seem to be largely a question of warning those with latent nasal infection, "chronic colds" and the like, to keep out of the water. Warnings of danger to the individual are far more impressive than warnings of danger to others; there should be an appeal to self interest. People with high narrow noses, occlusive deflections, or impinging middle turbinates, are all dangerous swimming risks. Persons with nasal discharges, "colds" and "catarrh," must be warned of their own danger by strikingly worded placards; let them stay out until they are well. Operative prophylaxis might include measures to free the drainage of all sinuses; infraction or partial resection of middle turbinates, submucous septal resections, removal of agger nasi or bullar ethmoid cells. Ostial occlusion by swollen mucosa should be made impossible. Heavy paraffin oil may be

dropped in the nose before swimming. Deep diving should be forbidden to all whose sinuses are large and ostia small.

It would seem that in harmony with the work of the Laboratory Section of the American Public Health Assn., this Academy might well advise the profession and the public regarding these safeguards and dangers of diving and swimming.

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DISCUSSION.

DR. ROSS HALL SKILLERN, Philadelphia, Pa.: There is no question that this is an important contribution to our knowledge of the etiology of sinus infection. All of us have seen cases of sinuitis following swimming, but I do not believe many of us have laid stress upon that point. I know I have not, although I have seen it time and time again.

One of the important points to be considered is that regarding the septic condition of the tanks. Personally, I think that has very little to do with it. I will not say it has nothing to do with it, but I believe only a low percentage of cases are due to this factor. I think the condition lies more particularly with the patient. If the patient has lowered resistance or some slight nasal infection, and then goes in swimming he is prone to get it. Take everybody that goes in swimming; if the septic tanks are the cause of these conditions, certainly we would expect that instead of one patient a week or one a month acquiring this sinuitis, we would have hundreds and hundreds, depending upon how many go in. I think it is a personal condition rather than a septic condition of the water.

Curiously enough, all of the cases that have come under my observation have been very acute. I presume the mild ones occur, but most of the ones I have seen have died. The very worst cases, fulminating, virulent cases, in two instances with brain abscess, have been directly traceable to swimming in a tank. Two of them were college boys, and

yet I was in communication with the college authorities and none of the other students were affected. One patient was from one college and one from another and were seen at widely different intervals.

I think Dr. Fenton's deductions are well conceived regarding the etiology. Swimming, particularly the deep diving, has much to do in bringing that process in some way, whatever it is, through the ostium into the sinuses. I have noticed it more particularly in deep divers than in those who swim on the surface. I think he goes too far in saying that those affected with this nasal difficulty should not swim. I would prohibit them from diving. They may go swimming or bathing if they like; then if they still show signs of trouble, it is time to come to his view.

I think that instead of using the heavy mentholated oil, the plugging of the nose with cotton before they go into the water,—not wool, because that can be washed out, but absorbent cotton—will prevent the water from coming in.

So in reviewing Dr. Fenton's presentation, I think we can say that swimming and deep diving adds a very important point to the etiology of acute, virulent sinusitis.

DR. EMIL MAYER, New York, N. Y.: I am very glad that Dr. Fenton brought this matter up, because I have had opportunity to see just this condition, that was so definitely traceable to the swimming tank, that there was no more question about it than if you saw somebody have whooping cough who had been in close touch with a patient with that disease.

I happen to have relatives in Nebraska. My niece lives in Norfolk, and I was much worried to hear that the child had a sinus infection. I learned that she was in the habit of swimming in a tank, and that every time after swimming, the attacks were much aggravated. I forbade any further swimming, although she was much disappointed. From the time she stopped swimming the attacks stopped, and when she came to me in New York she had no evidence of the trouble, although I had seen the X-ray plate and knew she had had antral disease.

There is a point that has not been brought out which is very important, and that is the character of the water. It is not soft water but very hard water that acts as an irritant to the nasal mucosa and produces this trouble. In my own bailiwick, I advise the patients to wash out the nasal cavity with warm salt solution, and do not specify what water they should use. I am sure that those who live in the middle West should add some soda to the water, so that they will have something to reduce the irritating effects.

It is a valuable thing for us to take home with us, to know that there is an etiologic factor. It recalls to my mind years ago, when we first got to learning many things about sinus disease, and I said that the automobile was responsible for many of these conditions. I had the satisfaction of seeing a headline in the morning paper stating that "A New York Specialist Finds Automobile Responsible for Sinus Infection," but notwithstanding this, I think anything that adds to our knowledge of the etiology of these conditions is of value.

DR. GEORGE W. BOOR, Chicago, Illinois: I feel very strongly on this subject, and do not agree with the two gentlemen who spoke. In Evanston we have Lake Michigan at our doors and also have two

swimming tanks. I am sure we see three times as much infection from the swimming tanks as from Lake Michigan. My own son was unfortunate enough to get an infection from swimming in the Y. M. C. A. tank, so I have reason to feel strongly.

DR. HARRY L. POLLOCK, Chicago, Illinois: This subject has been brought up particularly in regard to eye conditions. Dr. Gradle brought it up some years ago. In large cities we have an opportunity of seeing the infections. There is a swimming pool near our hospital, and several years ago I had three or four cases of frontal sinusitis from this one tank. At that time it was used constantly, and they did not have the constant intake and outlet of the water, but it was changed every twenty-four hours. All of these patients had the acute, virulent frontal sinusitis, and they had never had any trouble before. From information I gained from these patients, many others had the same difficulty. Since the plan has been changed and they have the constant flow of water, we have not heard of this particular thing happening.

While not related to the sinus, there is the large number of otitis infections that we also get in Chicago from the Lake. It is a large body, and when wind driven to the shore, we get much infection. We have every year many cases of acute otitis from diving and from the water. It is natural to suppose, that if a person has an infection, however mild, by diving in the water and going through the usual procedure of closing his nose and blowing hard, he will send the infection up into the sinuses. None of these patients that I refer to had ever had any infection until they had been in bathing, so it must come from the infected water supply. Hundreds of people go in, and we get only a few cases of infection, but this is explained by the fact that the others are healthy. If you expose a hundred children to scarlet fever, you will not get a hundred cases of fever as the result, but a few cases. I think the infection is due to water. All of the cases were quite severe, and they were all staphylococcic in origin. You can analyze the water from any public pool and find almost any bacteria.

DR. JOSEPH C. BECK, Chicago, Illinois: I wish to disagree entirely with Dr. Pollock. We have had many discussions on this subject, and I think the water has nothing to do with the infection, because of the changes which occur. I am sure if you leave your hand in the water for a few minutes, there is a noticeable action on the skin, and I believe that these people who go in swimming every day get a maceration of the skin of the auditory canal, and will get an infection as a result of this. The people who swim under water get these pathologic changes, and we have the changes in the tissues. I think it is the action of the water on the tissues which is detrimental, and that the water itself does not produce the infection.

DR. W. D. BLACK, St. Louis, Mo.: I believe this is a question where apparently both sides are correct, at least until the question is thoroughly settled by scientific investigation.

There is no question that the water in some of those tanks carry infection, as many of our cases have their origin in these swimming tanks: We also have cases that originate in places where the water is not infectious, as in a nearby mountain stream (the Merrimac River).

I have had many cases of infection of the ear, nose and throat, and I am positive that the water in this river is as sterile as in any stream.

It seems to me we have to take into consideration the fact that many swimmers expose themselves to cold winds and drafts and develop acute rhinitis, and often extension of the infection into the sinuses. We know that the nasal cavity contains bacteria at all times, and it seems to me that in these cases that occur from river swimming, any acute cold will bring on the attack, which is sometimes severe.

DR. RALPH A. FENTON, Portland, Oregon, (closing): I wish to thank the members for this very generous discussion, which is what I had hoped might occur. The paper was a humble effort to provoke discussion for the benefit of the rest of us.

We find that warning these patients not to dive does not work, because of the present style of swimming. People keep the head partially under water most of the time.

We investigated the mineral content of the water in our part of the country, where it is too low to have much effect, but there is an effect in waters containing chlorin.

If a committee could be appointed to work with committees from other associations it would be of help.

I am grateful to Dr. Beck for bringing to our attention the pathology that is produced by the water itself.

SKIAGRAPHIC STUDIES IN EAR DISEASE WITH RELATION TO THE DEGREE OF PNEUMATIZATION OF THE MASTOID.

NORVAL H. PIERCE, M.D.

CHICAGO.

The principal purpose of these skiagraphic studies is to ascertain the condition of the mastoid as regards its pneumatization, in a series of cases of hardness of hearing due to disease of the sound conducting apparatus, other than those with chronic suppuration.

The diagnosis was made by the usual functional tests, the Bezold-Edlemann continuous tone series being used, together with the Bezold large A for the Weber-Schwabach, (normal bone conduction 33 seconds), a small a^1 for the Rinné (normal bone conduction 16 seconds, aerial conduction 40 seconds), the Lucae c^4 for upper intensity and the Struychen's monochord for the upper tone limit. In making the Rinné test, the duration both of bone conduction and aerial audibility were recorded in seconds, a procedure which should always be carried out if we are to gain the full value of the test.

The first skiagraph is, perhaps, not intimately connected with the subject, as it is a case in which pneumatization is normal, but it exemplifies a class in which the principle features are increasing hardness of hearing due to stapes ankylosis with otoscopic evidence of caval inflammation.

A. M., female, aged 37, complains of hardness of hearing in the left ear, with a high pitched, ringing tinnitus. Cause unknown. Has noticed the defect of hearing for four years; it does not seem to be progressing lately. Complains of sensation of lightheadedness, but the description is not that of true vertigo of vestibular origin, and there is no nausea. Never had a discharge from ears. Father is hard of hearing. Had measles when a child.

Functional Tests: A. D. whisper; numerals 30 feet; medium pitched words 18 ft.; Weber not lateralized, 23 seconds. Rinné positive, (A.C. 35 seconds, O.C. 14 seconds). Low limit G_2 (24 d.v.). High limit b^6 . A.S. whisper; high and medium pitched words 5 to 6 inches; low pitched 2 inches. Rinné, negative, (A.C. 12 seconds, O.C. 15). Low limit, A, (55 d.v.);

high limit a^5 ; c^4 very much shortened. Turning to right, nystagmus, 8 seconds, vertigo 15 seconds. Turning to left, nystagmus 10 seconds, vertigo 20 seconds. The right tympanic membrane shows a light reflex reduced in length, lusterless, darkened, and the handle of the malleus is situated nearer the posterior periphery than is normal. The left mt. is slightly more retracted than the right, the light reflex is absent, the color is darker, it is lusterless, and the handle of the malleus is nearer the posterior periphery than normal. No improvement on inflation.

This is an instructive case. Obviously a disease of the left sound conducting apparatus, (fixation of the stapes) as witness loss of the low limit and negative Rinné, with involvement of the sound perceiving apparatus, as witness loss in normal bone conduction and the loss in the upper tone limit on the left side. The appearance of the tympanic membranes was indicative of the presence in the cavum of a plastic (catarrhal) inflammatory process. The position of the handle of the malleus is frequently seen in cases of arrested pneumatization. It is interesting to see by the skiagraph how extensively pneumatized the mastoids are. In view of the history of deafness in the family of the patient, we are inclined to regard the fixation of the stapes as due to otospongiosis, and not to the inflammatory processes, to which the hyperplastic mucous membrane which accompanies arrested pneumatization is subject. The appearance of the mt. indicates that tubotympanal inflammatory processes may occur in connection with normal pneumatization. However, the roentgenogram aids materially in forwarding the diagnosis and prognosis.

The second roentgenograms are from the mastoids of a female child, 8 years of age. They show complete (normal) pneumatization. This child has been under my care since she was thirty-two months old, at which time she had an acute suppurative otitis media, which ran a normal course and ended in recovery, although there was marked tenderness and some infiltration over the mastoid fossa in the first days of the disease. Since then she has had two additional attacks of acute otitis media, but of much less severity. Yet, we find that, although these three attacks of acute otitis media occurred during the period when the process of pneumatization of the mastoid was most active, they in no way interfered with its progress to extensive normal development. This is in accordance with the conclusion of Whittmaack. So long as

it is not interfered with before the beginning of the second year of life, pneumatization proceeds. That which causes arrest of pneumatization must occur in or before the first year, and that which occurs is not an acute inflammation, but a chronic latent process—the otitis media neonatorum. This is an example of a series of similar cases.

The third roentgenograms are from a female patient (A. H. 33-B-7), aged 22, who complained of recurrent attacks of earache and discharge since childhood with increasing hardness of hearing. The right tympanic membranes showed a diffuse light reflex, with fluid back of it in the cavum. No perforation. The left membrane showed areas of opacity and atrophy, with injection of blood vessels along the malleus; no perforation. The whispered voice was heard in the right ear 10 ft. for low pitched words, 18 ft. high pitched words; left ear, low pitched words 6 inches, high pitched words 12 feet. Weber-Schwabach to the left, 33 seconds. Low limit right ear D_1 (36 d.v.); left ear D_1 ; Rinné right ear O.C. 18; A.C. 27=+9 seconds. Left ear O.C. 26; A.C. 18=—8. High limit, right ear a^0 , left ear f^0 . Inflation brought hearing for whispered voice in both ears to over 20 ft., for both high and low pitched words. The roentgenograms show marked arrest of pneumatization in the right and total arrest in the left mastoid. Evidently the fixation of the stapes was not permanent. Nose, postnasal space and pharynx negative.

This case is typical of a large series which give a history of recurrent attacks of acute otitis media with discharge occurring since childhood, the discharge being purulent or more frequently mucoid or serous. The attacks in these cases are of short duration, but the hearing is most often increasingly impaired.

The fourth roentgenogram is from a female (E.M. 3-b), aged 64 years, with hardness of hearing in the left ear for 10 years, the cause unknown. There is slight pain recently, a crackling tinnitus in both ears. Her brother was hard of hearing. Never had a discharge. Membrana tympani right: areas of thickening (opacity), the light reflex is distorted and reduced in length and breadth, no perforation. Left: atrophic, light reflex is distorted and misplaced, no perforation. Functional tests: Whisper, right ear 20 ft., +. Left ear: low pitched words 10 ft., high pitched words 12 ft. Rinné right O.C. 11 sec., A. C. 25 sec., =+14; left ear O.C. 11 sec. A.C. 23 sec., =+12. Low limit right G_2 , left D_1 (36 d.v.). Weber-

Schwabach unilateralized, 32 seconds. High limit g⁶ in both ears. After inflation, the hearing for whisper in the left ear came up to 20 ft. for high pitched words, and low and medium pitched words 15 ft. Nose, postnasal spaces and pharynx negative.

The roentgenograms show the left mastoid to be unpneumatized in the posterior portion, only a few isolated cells showing in the region of the antrum, while the lower portion is diploetic. The left shows the same picture, except that the cells are fewer in number and are more obscure.

This is a case in a large series of tubotympanic catarrh. From the appearance of the roentgenograms we must conclude, in the light of Whittmaack's work, that the mucosa is thickened, and that the cavum is the seat of those pathologic changes which result from otitis media neonatorum.

I will not take up your time by exhibiting more pictures. Those presented represent many which have been taken in the past three years. The skiagraphic study of some hundreds of plates strengthens the opinion that Whittmaack's conclusions are in the main correct. We invariably find arrested pneumatization in chronic suppurative cases. But as you have seen, in those cases which present the picture of what we have called chronic catarrh of the middle ear, and tubotympanic catarrh with no history of chronic discharge, we also find arrested pneumatization in various degrees. Nor, is it a rarity to find more or less asymmetry between the two sides, the less pneumatized corresponding usually to the more affected ear as determined by functional tests.

For the purpose of description we may divide the mastoid into three types as shown by the Roentgen ray.

1. The completely (normal) pneumatized mastoid, that is a mastoid in which the cortex is thin and the cells occupy the whole mastoid process to the tip, backwards and upwards to the occipital suture or further, and above the antrum and cavum toward the zygomatic root. The cells are round and are systematically arranged, with the larger cells terminal. With such a picture, we may know that the mucosa lining the cavum and cells is normal—that is the epithelium cell is epithelioid—very thin and consisting of one or two layers. The subepithelial tissue is also very thin, homogenous, and cannot be separated into layers.

2. The mastoid in which pneumatization has been completely arrested. Here we may know that the mucosa in the

cavum and antrum is thick, the epithelium cuboid, with areas of ciliated epithelium; the subepithelial tissue is also thick and rather sodden; it may be separated into several layers, one of which may be identified as consisting of embryonic myxomatous tissue. It is richer in blood vessels, and this is infiltrated by round cells. In the cavum and antrum are abnormal strands and sheets of tissue, cysts and organized deposits.

3. Mastoids in which pneumatization is partially arrested. These may be divided into:

- a. Those in which we find the cells present only at the upper and posterior periphery of the antrum.
- b. Those in which the cells are present in this region and have spread downward between the external auditory canal and the sinus.
- c. Those in which pneumatization has proceeded so that the cells cover the sinus, leaving the posterior portion of the mastoid solid.

In all of these we have a thickened membrane such as we sketched under type 2., or fibrosis may have taken place in the thickened membrane, in which case the terminating line of the cell will be abrupt.

In order to appreciate the reason why the ascertainment of the condition of the mastoid as regards pneumatization is of scientific interest in this series of cases of nonsuppurative disease of the sound conducting mechanism, a knowledge of the work that has been done in recent years on normal and abnormal pneumatization of the temporal bone is necessary. To those who are unacquainted with this work, or to those whose memory of it has become dimmed, a brief outline of it needs no excuse.

Up to the recent past, three types of mastoids were regarded as normal, *id est*, (1) the wholly pneumatized mastoid, (2) the partially pneumatized mastoid, and (3) the sclerosed mastoids, or mastoids in which air cells were absent. Arthur Cheatele was the first to hint that the second variety was due to developmental irregularity, and referred to these mastoids as of an infantile type. The third type, the so-called "sclerosed" mastoid was believed to be frequently due to a sclerosing process, the results of chronic suppuration having obliterated previously existing pneumatic spaces. Whittmaack was the first to study the mastoid histologically in serial sections and in 1918, (?) published the results of his work of eleven years on normal and abnormal pneumatization of the temporal bone.

He divides the normal pneumatization of the temporal bone into three stages. In the first period, we have the formation of the cavum, recessus and antrum. At birth, these cavities are filled with myxomatous tissue. At first there is a mere slit existing between this myxomatous tissue and the tympanic membrane. The myxomatous tissue is covered with cuboid epithelium with its face to the tympanic membrane. Gradually, the embryonal tissue is absorbed or disappears, and the epithelium following it lines the air containing cavities; first the cavum, then the recessus and at last the antrum. Normally, when this pneumatization is undisturbed, the submucous tissue consists of a very thin layer over the bone, and the epithelium changes from the cuboid type, normal in the embryo, to the endothelioid epithelium, one or two cells in thickness.

At the end of the first or beginning of the second year, the second stage begins, and consists of the entrance of myxomatous tissue from the antrum into the marrow spaces of the underlying spongiosa. The marrow cells of these cavities are absorbed and replaced by the myxomatous tissue, and at a given time the epithelium from the antrum dips down, and as it dips down, the myxomatous tissue is in turn resorbed, until it becomes the submucous layer with the epithelium covering it. Thus an air space is made. This process goes on continuously by the invasion of marrow spaces by the myxomatous tissue from previously formed cells, until, consecutively, the air cells are completed at the end of the fourth or beginning of the fifth year.

The third stage extends through life, and is denominated by Whittmaack as the stage of interstitial pneumatization. This consists of the formation of small pneumatic spaces communicating with the larger spaces which were formed in early childhood, in the nodal points of the network which forms the walls of the preexisting pneumatic spaces.

His conclusions are, (1) the development of pneumatic systems in the temporal bone, that is, the tubal cells, the tympanic cells and the mastoid cells, follows a more fixed mode of developmental process with far greater regularity than has heretofore been believed. (2) The normal structure of the pneumatic process is characterized by a far reaching pneumatization and not by a sclerosing process. The individual peculiarities in the formation of the pneumatic system in later life, that is, after the fifth year, is not manifested by the

peculiarity of the mastoid as a whole, but only as an interference in the peripheral structure of the pneumatic system. It will be seen, therefore, that he recognizes but one type of normal mastoid and that is one in which complete pneumatization occurs. The presence of spongiosa is a sign of arrested development.

What is the cause of this arrested development? Here, perhaps, we have the corner stone of Whittmaack's deductions. In seeking for the cause of this disturbance of pneumatization, he reasons that the cause must primarily be searched for in the middle ear mucosa, the osseous structures evidently playing a secondary part in the process of pneumatization. Even in the normal process, it is quite evident that the bone is subordinate to the changes which are wrought in it by the submucous myxomatous tissue. It has been known for a long time, and written about by many authors, that the middle ear of the newborn and sucklings is subject to a peculiar inflammatory process, which has been denominated the otitis media of the newborn, otitis media concomitans, etc.

Schwartz, (Archiv für Ohren., Band 1, page 204) in 1864 recognized the "enormous frequency" with which inflammatory conditions are found in the middle ear postmortem in infancy, and in the description of the findings in one of his cases he says: "the middle ear is full of a thick, greenish-yellow pus, which contains well preserved ciliated epithelium. The covering of the middle ear is of a deep dark red color and is noticeably loosened. ***** *The ossicles are completely embedded in the swollen mucous membrane.*" There was no perforation in the tympanic membranes reported. This description tallies closely with that of Whittmaack, in describing the same condition as it occurred in his material. If I may digress for a moment, I would draw attention to the condition Schwartz found in the inner ear of these cases, as it throws a very suggestive light on the frequency with which we discover involvement of the sound perceiving apparatus in cases of arrested pneumatization in later life.

Von Troltsch also recognized the frequency with which otitis media occurred in infancy and young childhood. In a series of 24 postmortems on children, he found the condition in 17.

However, Schwartz yields the palm of priority for recognizing the frequent occurrence of what we now call otitis media neonatorum to du Verney, who is known for the

numerousness of his ear sections. In du Verney's "Tractus de Organo Auditus" (1684) page 36, he says: "Aperiui etiam complurium infantium aures, in quibus tympanum excrementis erat plenum, interim numquam, neque in cerebro, neque in osse petroso, inventa ulla prava dispositione."

It is latent in its character and course, and is discovered most frequently at postmortem table. According to Solow-zow, over 90 per cent. of all newborn children and infants are subject to this inflammatory process in the middle ear. Preysing, Görke, Rietschel, and, more lately, Göffert have extensively investigated this form of otitis media. The frequency with which it is found in children or infants rendered it difficult for Whittmaack to secure a large number of undoubtedly normal temporal bones in the first year of life. Much more easily could temporal bones with this inflammatory process of the mucous membrane be secured. He concludes that the process is not physiologic, but is undoubtedly a pathologic condition, and this view is supported by the presence of pus in the cavities of the middle ear and the known anatomic changes in the mucosa. These anatomic changes have been especially studied by Görke and Rietschel, and they agree that there are two definite types, a plastic and an exudative form, and Whittmaack suggests as a result of his investigations, that these may be divided into a latent, insidious type and a more acute form with a relatively rapid course. To these forms, Whittmaack adds the hyperplastic changes in the plastic otitis media, and an atrophic form with an exudate. It is evident that in the majority of cases we have a mixed type of these pathologic changes. The pure type of the first group is, however, clinically without symptoms and is, therefore, unsuspected during life. How this can affect the pneumatization will appear later.

The cause of this latent, symptomless, insidious form of inflammation was ascribed first by Aschoff to the aspiration of amniotic liquor, vernix or meconium into the tube and then into the cavum. The entrance of this irritant may occur in fetal life (aspiration of amniotic liquor), or during the time of birth (aspiration of amniotic liquor, vernix or meconium.) The pollution of the cavum with amniotic liquor is, according to Aschoff, the cause of the presence of leucocytes in the cavum of the newborn. According to this view, otitis media neonatorum is not an infectious process, but rather a reaction to a foreign body. It does, however, according to Hartmann

and other authors, render the structures more disposed to bacterial invasion. This is in agreement with the frequency with which bacteria are found in the exudate of the middle ear of newborn children. However, the foreign body reaction with sterile exudate may effect changes in the mucosa, which will interfere with or check pneumatization. In fact, Preysing in a great number of all ears which he examined in the newborn found an exudate which was sterile. Among the infected cases, the pneumococcus was by far the most frequently found. The changes wrought by the inflammatory process in the mucosa are characterized by a more or less intensive infiltration of the superficial layers of the mucosa and the epithelium with round cells, dilatation of the blood cells and production of an exudate containing more or less pus corpuscles. The epithelial stratum is changed to an extent that the ciliated epithelium (forming the tube) is found in places far removed, in the recessus and even in the antrum, areas in which the ciliated epithelium is never found in normal conditions. On this point, Whittmaack differs with Preysing and Görke. His conclusion, based on a study of a large amount of material, is that the extension of ciliated epithelium over the mucosa of the middle ear must be regarded as a pathologic process. He has proven by experiments on the lower animals—rabbits, cats, etc.,—by his production of a purulent inflammation in the ear, that he can produce a spreading of ciliated epithelium.

The normal epithelium of late fetal or early infantile life in the cavum is cuboidal in character, which changes under normal conditions to the epithelioid type. The persistence of the cuboidal epithelium and the presence of ciliated epithelium, Whittmaack regards as a mark of pathologic changes. The persistence of the cuboidal and ciliated epithelium coincides with other evidences of pathologic conditions in specimens showing interference with pneumatization, to a degree that suggests that his views are correct. Moreover, these abnormal epithelial types are not present in normal pneumatization.

Allowing for a relatively broad individual latitude in normal regression of the submucous tissues, there comes a time in the third to the fourth month where a normal type may be differentiated from an abnormal type. There can be no doubt, that in the presence of the inflammatory process under consideration, the superficial layers of the mucosa and the deep

subepithelial myxomatous tissue shows no tendency to regression but, on the contrary, displays a tendency to thicken. Whittmaack differs from Preysing in this regard and criticizes one of the illustrations which Preysing displays as a normal mucosa, as being evidently an abnormal condition dependent upon an inflammatory process. Whittmaack believes that a normally developed mucosa cannot be differentiated into various layers, and when this is possible it is due to disease.

We have, then, not only the persistence of more or less, of the embryonal subepithelial myxomatous tissue, but undoubtedly a proliferation of the same, an exquisitely hyperplastic mucosa rich in blood vessels. The superficial layer under the epithelium shows marked infiltration with round cells. Also there is a typical formation of lymph follicles. This thickening of the subepithelial tissue is especially to be found in the niches of the cavum and recessus. Another change which is characteristic of this condition, is the formation of the granulation nodules.

The point is made by Whittmaack, that this inflammatory process causes the total or partial arrest of the recession or disappearance of the myxomatous embryonal tissue. This does not occur in a regular manner over the entire surface, but in a very irregular manner, depending largely upon the inflammatory intensity at a given point. It can be readily understood, how bridges of membrane may thus be formed by an incomplete resorption, or abnormal strands of tissue, especially in the recessus epitympanicus and about the foot of the stapes—anywhere where there are sharp corners and depressions.

Granting that the inflammatory process causes persistence of the thick subepithelial portion of the mucosa, represented largely by a hyperplastic type derived from the former myxomatous tissue, and remembering the manner in which the epithelium follows the myxomatous tissue into the marrow spaces of the bone, we can readily understand how the process of pneumatization is arrested. It seems that the subepithelial tissue is deprived of its power of invading the marrow spaces, and where this does occur in a limited manner, the epithelium has no power of penetrating, but remains as a level layer on the thickened submucous tissue.

He concludes, first, divergences from the normal structure of the mastoid depend, without exception, upon typical processes, in consequence of changes in the character of the mucosa in the first and second years of life.

Second, the changes in the character of the mucosa may be grouped under the hyperplastic and fibrous types.

Third, the hyperplastic type develops from a latent, insidious plastic inflammatory process in the mucosa.

Fourth, the fibrous type depends on an acute exudative inflammatory process.

Fifth, whether disturbance of the pneumatization is partial or complete depends upon the intensity of the changes in the mucosa.

Sixth, every type of disturbance of pneumatization gives a typical structure picture of the mastoid—

I. Complete arrest of pneumatization.

a. By hyperplastic.

b. By fibrous mucosa.

II. Partial arrest of pneumatization.

a. In the hyperplastic inflammation (severe, intermediate and light grade.)

b. By fibrous mucosa (prolonged pneumatization).

Seventh, the concurrence of hyperplastic with fibrous changes in the mucosa occurs with relative frequency, and leads to mixed forms of structure types, sometimes with one and sometimes with the other component predominating.

What relationship does pathologic pneumatization bear to changes in the tympanic membrane? As the tympanic membrane is the one visible portion of the otic structures, it is interesting and important to know whether the changes in this membrane bear any relationship to pathologic pneumatization. Whittmaack is unable to say at the present time just what relationship slight and moderate anomalies in the tympanic membrane bear to the pneumatization process. This is readily understood when we remember that changes occur rapidly in the tympanic membrane after death. He believes, however, that he is safe in saying that the manubrium in the otoscopic examination lies in a course that is nearer to the posterior external auditory canal than in normal cases, and the circumference of the tympanic membrane appears more oval and is smaller than normal in cases of disturbance of pneumatization. In other words, the infantile type of tympanic membrane persists. Changes in the tympanic membranes themselves may or may not have a relationship to the character of the mucosa within the cavum. He points out, however, that slight diffuse and regular cloudiness of the tympanic membrane, with a diminution in the luster, is an

indication of disturbance of pneumatization, together with the other changes in the mucosa which accompanies these disturbances. This cloudiness is due to increase in the cuticle layer of the tympanic membrane, and the decreased luster is due to a greater desquamation of the superficial layers. The cuticle layer of an entirely normal adult consists of a single layer of perfectly flat epithelium, and this gives a bright luster. In hyperplastic changes of the mucosa, we often find the deep cuticle layers composed of cuboid cells. The subepithelial layers show a thicker and richer vascularity. The superficial layers of epithelium are raised in lamellae, which give them a rougher surface. This gives the cloudiness and lack of luster of the tympanic membrane on otoscopy. This is a normal condition in the first period of development, and reaches frequently into the second period. In this way it is explained why in sucklings and small children, we seldom find a tympanic membrane with the luster of the adult tympanic membrane. We very frequently find, by otoscopy, atrophy of the tympanic membrane in connection with the fibrous changes in the mucous membrane, with complete or marked arrest of pneumatization. This is due to the arrest of the *membrana substantia propria*.

Abnormalities in the tympanic membrane are associated with arrested pneumatization, indicated by spotting of the tympanic membrane by areas of thickening associated with areas of atrophy; the areas of thickening appearing as whitened islands, while the areas of atrophy give a darker tone, due to their translucence and the color of the membrane within the tympanic cavity.

Calcareous deposits in the tympanic membrane are not associated with changes in pneumatization. Peripheral cloudiness of the tympanic membrane is frequently associated with changes in the mucosa accompanying pathologic pneumatization.

What is the relation of pathologic pneumatization to the accessory mucous membrane bands and folds? It is known that in the *recessus epitympanicus* is found a number of ligaments and duplicatures of the mucosa. Whittmaack attempts to answer the question: What is the absolutely normal anatomic content of the epitympanic space? If we confine ourselves to the temporal bone with a completely undisturbed development, we can say that in the recessus we find besides the two folds, the ligamentum mallei externum sive laterale,

the ligamentum mallei superior and the ligamentum mallei anticum. Occasionally one or the other of these ligaments is lacking. Especially is the ligamentum mallei superior inconstant. It is absent with relative frequency in high tegmens and well developed pneumatic cells.

When the bones are examined at a time when the myxomatous tissue is not yet completely absorbed, the anlage of these ligaments can be discerned within the tissue in the form of fibrillary strands, and presents a resistance to the ingrowing epithelium similar to that displayed by the ossicles.

The development of the inconstant so-called accessory folds frequently occurs from the incomplete absorption of the sub-epithelial tissue. In this way small bridges of tissue or sheets of tissue originates, reaching from one wall to the other of the epitympanic space. Zuckerkandl has previously described the development of accessory folds in a similar manner.

The arrest of pneumatization in the mastoid has a tendency to draw the sigmoid sinus forward, a very practical fact to remember in our operations on such cases. Especially noteworthy, is the protocol in one of his cases (No. 38), in which complete arrest of pneumatization is pictured, and where the displacement forward of the sigmoid sinus is most marked.

In summing up the relationship which arrested pneumatization bears to other parts of the temporal bone we find:

1. There undoubtedly exists a certain relationship between pathologic pneumatization and certain anomalies of the tympanic membrane, lusterlessness, cloudiness, atrophies, scars, etc.

2. Changes in the tympanic membrane cannot be regarded as always constant accompaniments of pathologic pneumatization.

3. In entirely normal pneumatized temporal bones, we find only the constant ligaments in the epitympanic space. The development of accessory folds is a sign of pathologic pneumatization.

4. The displacement forward of the sigmoid sinus is found in pathologic pneumatization, the higher grades of displacement only with the extreme types of disturbance of pneumatization.

5. The persistence and unusual breadth of the fissures is an accompaniment of pathologic pneumatization.

It is apparent that the work of Whittmaack explains many hitherto unexplained problems. It explains, for instance, the so-called chronic catarrhal otitis media which arises from apparently no cause, and which has been explained on the hypothesis of a diathesis or a catarrhal inflammation. It explains the chronic tubotympanic inflammation, or at least places these conditions in an entirely new light.

The more important conclusions as regard the relation of arrested pneumatization to inflammatory disease of the ear may be summed up as follows:

1. Practically all severe forms of suppurative middle ear involvement develop in temporal bones with pathologic pneumatization, insofar as this depends on continuous extension from the tube.

- II. Chronic middle ear suppuration exists on the basis of complete arrested pneumatization, or the severest forms of disturbance of pneumatization, with markedly hyperplastic mucosa, and develops in the child;

- A. Chronic suppuration of the mucosa on the ground of the acute exacerbations of the suckling.

- B. The chronic cholesteatoma suppuration, either (a) after acute necrosing otitis through ingrowth of the epithelium, in consequence of large peripheral defects of the tympanic membrane, or (b) as an insidious process with intake of the *pars tensa* after sequestration of the antrum recessus from the cavotubal cavity, in consequence of adhesions from Schrapnell's membrane, or an atrophy above, or adhesions above or below the posterior folds; (c) middle ear suppuration, with epithelization or combination recessus cavum and cavum cholesteatoma suppuration, and a combination of the process which leads to cholesteatoma formation.

- III. The form which the chronic suppuration and its course pursues is preordained by the anatomic changes within the several cavities of the middle ear, before the appearance of the clinical symptoms. Also, the secondary and end processes, such as the extent of perforation, epidermization, polyp formation, scarring, etc., depend upon a preformed anatomic substratum.

- IV. The acute middle ear suppurations develop with predilection for medium and lighter grades of disturbance of mucosa and corresponding character of the mucous membrane.

- V. The greater the hyperplasia, the thicker the epithelium, and the less the pneumatization, the greater is the tend-

ency toward the occurrence of acute inflammatory process and eventually a protracted course, and the less, on the contrary, is the tendency to frank mastoiditis and vice versa.

VI. The character of the secretion in an acute otitis media stands in direct relationship to the character of the mucosa. Thick, highly hyperplastic mucosa with ciliated epithelium is especially apt to produce a mucous or mucopurulent secretion. Slight hyperplasia with flat epithelium gives thick, purulent, tenacious secretions. Fibrous changes predisposes to a thin fluid, serous or seropurulent secretion.

VII. Normally pneumatized temporal bones are most frequently infected in acute middle ear disease, with resulting frank mastoiditis.

VIII. Middle ear inflammations of tubercular and luetic character exist on the anatomic substrata of the mucous membrane and its accompanying disturbance of pneumatization. Their course depends also on the changes in character of the mucosa as it occurs in the course of pneumatization.

The relation which pathologic pneumatization bears to endocranial complications is most interesting and important. In the pictures which I shall cause to be thrown on the screen, you will find how frequently abnormal vascular communications persist between the abnormal mucosa, the meninges, the bulb of the jugular and the sigmoid sinus.

It would be impossible to give a complete account of the monumental work performed by Whittmaack. We must be content with this short and incomplete sketch.

Before closing, I must, however, accentuate the very kernel of Whittmaack's deductions, namely, that nearly all inflammatory diseases of the middle ear, in their genesis, nature and course are dependent on certain anatomico-developmental changes in structure of the mucosa and osseous structures of the ear. Most of these alterations in structure are caused by a latent, insidious, inflammatory process, which occurs in early life. In other words, if in late life an individual develops middle ear inflammation (catarrhal) with adhesions, fixation of the stapes, etc., the special suppurative type of inflammation is predestined, when the occasional cause arrives, by the changes which have occurred in the first years of that individual's infancy. On this fundamental principle, he has erected a plausible, logical structure, which must be proved or disproved by future investigations. True, there are discrepancies, and here and there we discover findings and conclusions which are susceptible of quite different interpretations,

but on the other hand he throws light on many dark corners of otology, and explains in a logical manner many points of pathogenesis which have heretofore been merely surrounded with meaningless words.

DISCUSSION.

DR. G. W. MACKENZIE, Philadelphia, Pa.: I accept it as a compliment to have been asked to discuss Dr. Pierce's talk on so important a subject as the "Normal and Pathologic Pneumatization of the Temporal Bone." The subject is so large and the time so limited, that I find it beyond my ability to do justice to the subject, or to Dr. Pierce who has so ably presented it.

Much that I would say would necessarily be a repetition of what has already been said; however, if I can but help somewhat to fix more firmly in the minds of some present the points already brought out by the essayist, your time will not have been wasted.

For the greater part of our present-day knowledge, we are indebted to Professor Karl Whittmaack, who has made exhaustive researches covering a period of several years. No one of whom I know at this time is prepared to refute either the work or conclusions of Whittmaack. In his Atlas of III Figs., drawn from Microscopic Sections, he illustrates the normal process of resorption of the embryonal myxomatous tissue that is present in the preformed middle ear spaces and mastoid antrum; following these he presents another series showing the abnormal.

In comparing his normal series with my own collection, I find agreement in every detail. Concerning the pathologic series, my collection is so small compared to his, that I am in no position to tab up with him, and for want of proof to the contrary, must accept his presentations and conclusions. If one follows Whittmaack's pathologic theories, he is forced to accept the same conclusions as the author.

The normal series, beginning with the fifth to sixth month of fetal life, shows at this earliest period the presence of a narrow epithelial lined lumen in the external part of the preformed tympanic cavity, i. e. directly behind the membrane. The bulk of the cavity, as well as the antrum, is filled with embryonal myxomatous tissue. This lumen gradually increases in size up to the ninth month of fetal life, when the epithelial lined cavity occupies approximately one-third of the whole preformed tympanic cavities, the remaining two-thirds being occupied by embryonal tissue. Shortly after birth, the lumen increases appreciably in size, especially toward the epi- and hypotympanum, where there still remains a considerable cushion of subepithelial embryonal tissue. This process of pneumatization continues progressively throughout life, when not interrupted by any pathologic state; so, too, does the advance of the myxomatous tissue into the spaces of the surrounding bony walls, except where it is prevented by the more compact bone of the osseous labyrinth and facial canal.

In the event of an intercurrent attack of nonsuppurative otitis during the nursing period (which is not so uncommon as one might think), the further progress of pneumatization is arrested. The subepithelial cushion remains as it is. The resulting exudate in the tympanum or-

ganizes at various points, where the epithelium is lost. From the subepithelial embryonal tissue, blood vessels sprout forth into the exudate, so that in a well developed case of its kind, there will appear many small, isolated, epithelial lined spaces, together with the more central larger one. In some instances the smaller spaces appear in a rosary like series, more often seen in the epitympanum. When the exudate forms on the median surface of the tympanic membrane, this same process of granulation with later organization occurs as illustrated in Figure 31 of Whittmaack's series.

From the thickness and location of the cushion of subepithelial tissue present in a pathologic case examined in later life, one is able to judge approximately the age at which infantile otitis with resulting hyperplasia had begun. Generally speaking, the thicker the pad, especially in the epitympanum and the antrum, the earlier in life the otitis occurred, and vice versa.

With the advent of infantile otitis, there is not only an arrest of the normal progress of pneumatization, with a remaining thick subepithelial cushion, but there occurs at the same time an arrest of the advancement or the further development of an invasion of the embryonal connective tissue into the surrounding bony walls, which occurs under normal conditions.

There is one thing which Whittmaack mentions that contradicts the teaching of pathologists up to this time; for instance, in Figure 87, he shows a section of the middle ear epithelial lining, in which there is a transition from the flat epithelial over to the ciliated type. I do not wish to deny the possibility of metaplasia from the lower order of cells to a higher order, but we have, up to now, generally accepted the view that metamorphosis is rather in an opposite direction, that is, from a higher to a lower order. It is impossible, as I said in the beginning, to do the subject justice in a few minutes' discussion. The subject is large and important—important in that if all that Whittmaack claims is true, it marks an epoch in aural pathology. I hope that Dr. Pierce may be able to spare the time to translate the work into English.

DR. JOS. D. HEITGER, Louisville, Ky.: The practical importance of this work will appeal to you when you go back to your text books and realize that we have been accustomed to speak of the types of mastoid as diploic, pneumatic and mixed. The changes brought about by the myxomatous tissue produce an arrest of pneumatization, and two of the so-called normal types, namely, the mixed and diploic, are really abnormal, and the one type that Whittmaack considers normal is the completely pneumatized temporal bone.

Another point of importance, which Dr. Pierce mentioned in closing, is that we have formerly considered sclerosed bone as an end process of chronic suppuration. We have to revise that view in the light of this pathology, and consider the sclerosed bone as the beginning and this long drawn out suppuration as the end.

The next practical point that comes to my mind is, that in the individual who in later life develops a mastoiditis, the character and course of his trouble have been predetermined, one may say, by the pneumatization, either arrested, delayed or complete, which occurred during the two first years of the life of that individual. Dr. Pierce mentioned

the fact that about ninety per cent. of infants are afflicted with this lowgrade inflammation or irritation of the lining membrane of the middle ear. With figures as high as this, it is a wonder that any of us escape chronic suppuration and mastoiditis.

There is one thing that I think has not been mentioned in regard to the clinical course of this infantile middle ear disturbance, which Whittmaack speaks of. He divides this pathologic condition into two types, the hyperplastic or thick type and the atrophic or thin type, and he claims that these two types run a different course, and are even characterized by having different types of secretion. He mentions some changes in the development of the drum membrane, the ossicles, and the contents of the epitympanic space, as well as the position of the lateral sinus, but these are more or less minor, and he draws no definite conclusions from them. He lays a great deal of stress on the vascular connections with the meninges, which render these types more liable to intracranial complications.

Mygind, in a review of a thousand mastoid operations, in a recent issue of the *Acta Oto-Laryngologica*, corroborates Whittmaack's findings, and it is an interesting fact that the keenest critics have not been able to disprove his findings and most of his conclusions.

DR. JOSEPH C. BECK, Chicago, Illinois: I am sure we all appreciate Dr. Pierce's presentation. It is not the first time I have had the pleasure of discussing this work, but I enjoy looking at these pictures every time I see them. I have them at home and often look at them.

I wish to emphasize the point brought out by Dr. Mackenzie. Since the book is in German, it requires translation. I talked of this with Dr. Pierce, but he did not take kindly to it, so Dr. Bigelow, of Providence, and I got together and communicated with Dr. Whittmaack, who gave us permission to use the cuts and put the work in English. He was kind enough to say that he would be very glad to have the work translated, and would lend every possible assistance. I am hoping that Dr. Pierce and Dr. Bigelow will get together on the work and put it into English, so that we may all have full benefit of it.

We must remember the great difference that the X-ray makes in the diagnosis. We speak of having a sclerosis or a fibrosis of the mastoid. Dr. Pierce has presented a number of radiographs before the Chicago Laryngological and Otological Society, showing that there is a great preponderance of obliteration of the mastoid cells, or so-called normal diploic type of mastoid. I do not accept all of this. I have some specimens that modify them. A patient of mine had a complete mastoiditis picture showing pneumatization, but eight years later she returned from China with a reversal of this condition. That is only one case, and it does not speak against all this work, but there is here the likelihood of fibrosis.

The point was brought up by Dr. Barnhill as to what this condition has to do with disease, but according to Whittmaack it tends to produce disease.

DR. J. M. INGERSOLL, Cleveland, Ohio: I have always thought that the chronic suppuration in the mastoid was a factor in producing the sclerosis, and that the sclerotic bone was the result of the suppuration. I have had a few cases of chronic suppurative otitis media, such as Dr.

Beck mentions, in which X-ray pictures were taken after an interval of three or four years, and in these cases the sclerotic process seemed to have advanced.

The statement that 90 per cent of the newborn have some inflammation in the middle ear is surprising to me. In nearly all severe infections, it is not uncommon to find in the lungs and in the sinuses of the head some symptoms of accumulation of secretion, and in such cases post-mortem examinations often show secretion in some or all of the accessory sinuses of the nose. I wonder if the diseases which causes the death of the children, upon whom the postmortem examinations were made, were not the cause of the secretion accumulating in the middle ear.

DR. GEORGE W. BOOT, Chicago, Illinois: There is no denying that Whittmaack has done a beautiful piece of work, but why swallow it hook, line and sinker? I had a patient over seventy years of age with a mastoiditis. According to this theory, it was because he had defective pneumatization, and he had this defective pneumatization because he swallowed amniotic fluid during intrauterine life. What is this but predestination?

DR. NORVAL H. PIERCE, Chicago, Illinois, (closing): It is pretty hard to tell just where to begin in attempting to close a discussion on this subject.

First, is it possible for a chronic suppurative process of the mastoid to produce obliteration of these normal pneumatic spaces? I do not know. We know that it happens in other places. It happens in the internal ear, where the semicircular canals will be completely obliterated by dense, hard bone, but up to the present time we have yet to see microscopic evidence of preexisting pneumatic spaces being changed into bone. It is altogether different from the osteitis of the long bones. There we do have a sclerotic condition occurring in bone that is without air spaces, but in order to obliterate a pneumatic space, you have first to obliterate epithelium, and epithelium is very difficult to obliterate. You may get rid of some of it, but if any remains you will have a space. That is why it is so difficult to cure some cases of mastoiditis in children. If you ream out everything down to the inner tablet of the skull, there is a very strong likelihood of that child having a so-called recurrent mastoiditis. Everytime a child takes cold, the mastoid swells up, fluctuates and there is trouble. Why? Because the epithelium from the cavum will spread over the surface of the bone before the bone cells can form bone. In consequence, we have a large air space (antrum) reformed, which is in very close proximity to the surface, and you have an abscess. Up to the present time, nothing has been presented to the otologic world on this subject that disproves this work.

A SUBMUCOUS PLASTIC OPERATION FOR NASAL DEFORMITIES OF CARTILAGINOUS ORIGIN.

J. M. BANISTER, A.B., F.A.C.S.

OMAHA, NEBRASKA.

This being a practical surgical paper, and one purely clinical in scope, no time will be allotted to an academic discussion of nasal deformities.

Only one special form will be considered, namely that type, in which the septal cartilage has been displaced laterally, with dislocation of its tip and anterior margin from their position behind the columnar cartilage into either nostril. As a result, there is very commonly a lateral deformity of the nose with depression of the nasal tip, to which must be added the consequent obstruction and interference with function.

Deformities of this description, as we all know, have long been bugbears to the specialist. An ordinary submucous resection would secure a lessening of the obstruction, it is true, but would increase the deformity, and would not be advisable in childhood. An operation, therefore, of the plastic type, which while permitting the preservation of the septal cartilage, will relieve the lateral deformity and elevate the depressed nasal tip, and at the same time remove the intranasal obstruction caused by the displaced cartilage, can be said to be of real practical value.

Dr. Oliver Tydings of Chicago, in a paper read before this Academy in 1914, brought forward a new submucous septal operation, and has elaborated his method in other papers more recently. I was not aware of Dr. Tydings' work in this direction until very recently, when I received from him several reprints of his papers, so that the priority of his procedures in this regard is thoroughly established. However, the method which I shall present in this paper was independently evolved and practiced before my learning of Dr. Tydings' operation and technic. The two methods will be found to differ quite materially in certain regards. In 1920, Dr. T. E. Oertel, of Augusta, Georgia, presented before the Boston meeting of the American Laryngological, Rhinological, and Otological Society a paper entitled "Submucous Replacement for External Deviation."

From a perusal of this interesting paper, I was impressed with the value of the swinging septal flap, and proceeded to make use of this feature of Oertel's method in an operation, which I had to do at that time. Otherwise, the technic of my operation is very different from his, as can be seen from a comparison of the two operations as described. It should be borne in mind that, as before stated, the operation now to be described applies only to the relief of deformity resulting from lateral displacement of the septal cartilage, with dislocation of the anterior margin of the same, and has no reference to conditions due to deflections of bone. The technic of the method is as follows:

An incision is made along the anterior margin of the dislocated cartilage, as it presents just within the nostril on the side involved. This incision extends from the extreme upper limit of the margin to the lowest point of its anterior aspect, and divides the mucosa and perichondrium down to the cartilage proper. The soft parts are then elevated toward the columnar cartilage, that is toward the median line of the nose, until the anterior margin of the dislocated septal cartilage is well cleared on that side. This furnishes a cul-de-sac behind the columnar cartilage for the tip and anterior margin, when the septal cartilage is finally brought into its proper lineal position.

Attention is next directed to the opposite border of the incision along the anterior margin, and from this point the soft parts are thoroughly elevated, as in the ordinary submucous resection, the separation being carried well back over the perpendicular plate of the ethmoid. An incision is now made through the septal cartilage, beneath the elevated flap of the soft parts, just in front of its line of junction with the perpendicular plate of the ethmoid, and this incision carried from the extreme upper limit of the cartilage down to its lower margin. This division should be strictly limited to the cartilage, the uncut soft parts on the other side of the septum being elevated backward, through the incision, for about a quarter of an inch, to permit of proper motility. Then the lower border of the triangular cartilage, which as described, has been freed anteriorly and posteriorly, is thoroughly separated from all attachments, commencing at the antero-inferior angle and continuing the separation back to the lower extremity of the posterior vertical incision, without opening the soft parts between this margin and the opposite nasal cavity, the perichondrium being simply elevated along the op-

posite, or inner, side of this freed lower border to permit of the reduction of the cartilaginous flap into its proper position in the median line. No attention is paid to the vomer or its coverings. When the indications, as described, have been fulfilled, a swinging flap of septal cartilage will have been formed, the hinged margin being along the nasal crest, and the three other margins free and moveable. It can thus be seen that the perichondrium and mucosa are left undisturbed on the one side with the exception of the narrow margins separated, as noted, for securing greater motility. Furthermore, the entire operation is performed submucously through the one incision along the anterior margin of the dislocated cartilage. By introducing an index finger into each nostril and

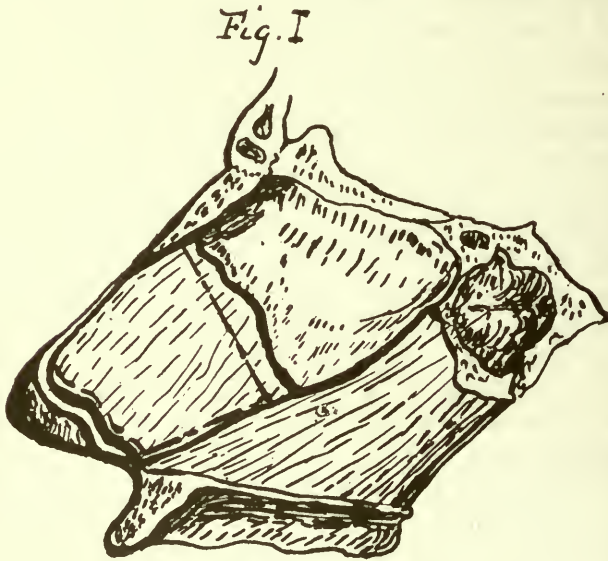


Fig. 1.

elevating the nose somewhat by their means, the swinging cartilaginous flap, still covered on one side by the mucosa and perichondrium, can be reduced between the two fingers into its proper position in the middle line, the lateral deformity being relieved, the elevation of the nasal tip restored, the symmetry of the nostrils secured, and the breathing space on both sides correspondingly amplified at the same time without loss of tissue. Two sutures uniting the margins of the anterior incision are passed, thus anchoring the tip and anterior margin of the cartilage in the proper position behind



Fig. 2.



Fig. 3.

the columnar cartilage. Kyle's perforated intranasal splints are introduced, one on each side, and two crossed strips of adhesive plaster, externally, complete the dressing, and furnish the entire support. No pins or external splints are used. There is no spring to the cartilaginous flap, and it readily remains in place.

In certain cases of the type of dislocation of the triangular cartilage referred to, there is a very decided knuckle, or bend, of the cartilage in the vestibule on the side opposite to that into which the anterior margin is dislocated. Under such conditions the operation is performed in exactly the same way as that described, with this modification.

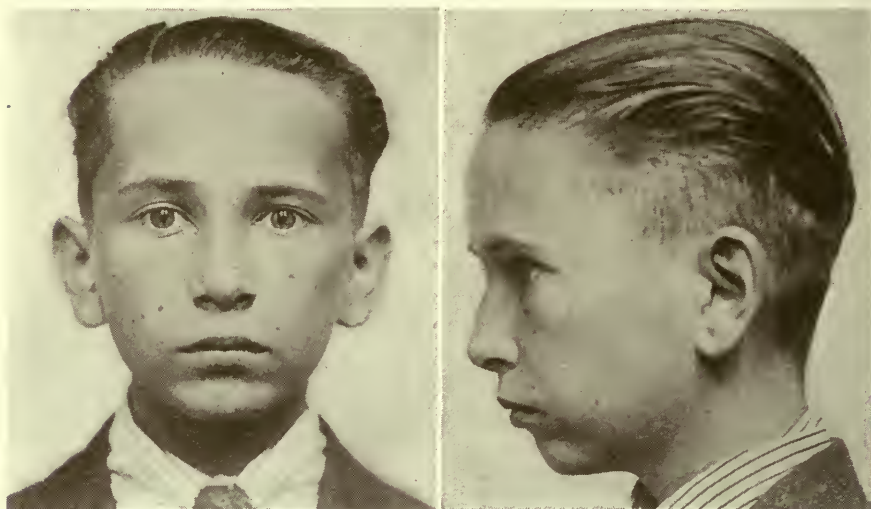


Fig. 4.

After completing the elevation of the perichondrium and mucosa, as described, the vertical incision of the septal cartilage is first made just at the angle of the knuckle, on its concave side, under the flap of the elevated soft tissues, of course, and a second incision is placed parallel with this farther back and just in front of the perpendicular plate of the ethmoid. We, thus, have *two* parallel vertical incisions through the cartilage instead of one, and there are formed two swinging cartilaginous flaps at the conclusion of the operation. These two flaps swing into position quite as readily as the one, and while there may be, in extreme cases, some slight overlapping of their margins, this does not complicate matters or interfere with the success of the operation.

A great point in favor of this operation is that it is applicable to deformities of the type under discussion occurring in children. This advantage need not be dwelt upon before the present audience.

In Figure 1, a diagrammatic representation of the anatomy of the swinging flap is given, the dotted lines showing the location of the incisions.

Figures 2 and 3 are postoperative photographs, in cases in which there was marked deformity of the lateral type, with dropping of the nasal tip.

Figure 4 shows postoperative photographs in the case of a boy, thirteen years of age, where the lateral deformity was extreme, and where there was the septal knuckle referred to, extending so far into the opposite cavity, as to reach the inferior turbinate and entirely block the breathing space.

In this case, I performed the operation with the twin incisions, which secured the result shown, and at the same time removed the obstruction to breathing without the sacrifice of a particle of tissue.

The operation brought forward in this paper is easily performed with the instruments commonly used in the ordinary submucous resection, not a single special instrument being required. The technic described is presented simply as a surgical suggestion, and with no claim to its being a panacea. It has been useful in my hands, and may possibly be of assistance to others in these trying cases.

DISCUSSION.

DR. F. J. PRATT, Minneapolis, Minn.: Two years ago, at the meeting in Denver, Dr. Banister explained the technic of this operation to me. It looked good, and I intended to do it, but for some reason have not tried it. I did not suppose until to-day that it would take care of any large deformity, but thought it was only for the small ones. Dr. Banister, however, tells me that it takes care of good large ones. It is like the old Gleason operation in principle, except that the Doctor's is under the membrane. I can see where it would do very nicely in children, especially the ones who are too young for a submucous resection, because it will not only give them a breathing space, but will also lift the tip of the nose.

DR. OLIVER TYDINGS, Chicago, Illinois: A case was brought to me in a child less than ten years of age. Realizing what this deflection would mean to the child, I worked out this operation. I am sorry that Dr. Banister limited his operation to the cartilaginous septum. It is not so limited, but can be used on the bony septum. I do not make my incision quite as large as his, for you so frequently have deflections of the bony septum in connection with the cartilaginous septum. I go

through here (illustrating on board), travel along the line of least resistance to the vomer, and go down as far as the depth of the deflection, until I get to the interlacing fibers of the vomer, and then I take the cross dissector of Freer's (I use all of Freer's instruments in this work) and go behind in this way (indicating). If you undertake to make this incision from before backward, you will be almost sure to get a perforation, but if you do it in this way, you can cut down to the floor of the nose and never get a perforation. Then I go through and in, coming down on the vomer after I cut these fibers; then I come parallel to that same instrument, and sever the cartilaginous portion below from its dislocated position. If you have a redundancy of bone, remove it. If not, let it alone. Many of these cases can be straightened, and when you do so, you will find that it will become perfectly straight in the nose. After you have done them in this way with the bone, you scarcely need anything, for it will remain perfectly straight, but in some instances you may have to use one thing or another in the way of a splint in the cartilaginous cases. For the purpose of keeping the cartilage straight, I have found the old-fashioned shawl pin, about two inches long, to be the best thing. You can go under the mucosa from the convex side and fasten the pin down into the maxillary crest, and you can fix it so that the person has plenty of breathing room. You can keep it there from two to ten days. I have had two cases, one in a physician, who have worn them for six weeks. This patient wrote and thanked me for the courtesy, and said the pin had been lost. I told him to have an X-ray picture made and he would find it. About two years ago, a lady consulted me and said there was something the matter with her nose, that it felt as if there was something in it. I examined the nose and there was my pin, which I had put in place several months before. I had told her to come back after a few days, but she did not do so because the nose felt all right. I was not willing to risk my reputation for veracity on a statement of that kind, so I brought in the members of the office and showed the case to them.

If you will try this operation, you will find it the quickest and most satisfactory operation that can be done upon the septum.

DR. JOHN F. BARNHILL, Indianapolis, Indiana: I do not wish to discuss the paper or operation. I wish only to discuss a remark that the author of the paper and others have made. I refer to the reluctance of most rhinologists, and the advice of most books and articles, about performing a submucous resection of the nasal septum in children. It seems to me this is a question we should settle more definitely than it has been settled. Following the advice of teachers and writers, for a long time I felt that such operations on children were inadvisable, that they might result in much deformity, and that for this reason we should not do operations for the correction of deformed septa in little folks. However, we find many young children who need operations as badly as adults, and it is probable that the development of the child is as impaired because of nasal deformity and nasal stoppage, as it would be were the impaired breathing due to enlarged tonsils or adenoid, and we would, therefore, be justified in operating and giving them air. So, on those grounds, I have operated on little children, and

have had no bad results of any kind; hence the query, do we advise against operations merely because of the difficulty of doing them, or is it because deformity actually has been observed in well operated cases? It is not easy to operate on children; we must have a general anesthetic, and there is annoying bleeding, but is there any other real objection to submucous resection of the nasal septum in children? I am convinced there is not.

DR. JOSEPH C. BECK, Chicago, Illinois: I object to operating on children, because I have seen the effects of operation in cases referred to me for correction after an operation had been done.

Dr. Banister should have qualified the type of operation. I have recently operated upon a boy of seven or eight, and it is a graduated operation. We know that up to at least twelve years of age, the areas of ossification and development of bone and cartilage are in question, and any interference at the junction of these points by operation is going to make a different nose than that head should have.

DR. J. M. BANISTER, Omaha, Nebraska (closing): This subject has been brought forward to create discussion, and to give the essayist the views of other members of the Academy upon the method advocated. That object has been attained, and further discussion on the part of the writer would seem unnecessary.

A NEW METHOD TO DEMONSTRATE AND TEACH THE SURGICAL ANATOMY OF THE NOSE AND ACCESSORY SINUSES.

ROBERT F. RIDPATH, M.D.

PHILADELPHIA, PA.

Many times, in the teaching of this subject at the Post Graduate School of the University of Pennsylvania, have I been at a loss to illustrate many of the points, which in a course on this subject presents itself from time to time, in spite of the fact of possessing numerous manikins, models, diagrams, illustrations, lantern slides, etc. We are all aware that the various models which are used for teaching and illustrating, do not, in the proper sense, apply to many phases of the subject. We are also aware of the lack of interest and false impressions which may be given by the usual flat drawings, no matter how accurate these may be. It is utterly impossible for the student to carry away from the lecture room an impression, permanently imprinted on his mind, by which he may recall without an instant's hesitation any anatomic relation or structure he may for the moment wish.

Most of those teaching may feel that he has made an indelible impression by his blackboard drawings, when, if he should ask nine out of ten of his students to go to the board and illustrate the point one week later, failure would result.

It was these numerous failures, oft repeated, which finally convinced me of the fact, that the fault must lie not so much in what was said, as to what and how the fact was impressed on the mind. In dealing with students, in spite of the fact that these may be postgraduate students, we must always remember to take an illustration from a battleship formation as our guide, and never forget that the rate of speed is only as fast as the slowest vessel can travel.

If, therefore, we have a means whereby the student is impressed to the degree of always remembering, we have advanced. It is a true saying that "what the eye perceives the mind retains."

It, therefore, is my privilege to present to you a method, that although I shall illustrate only a few points, the hope is that these may be sufficient to demonstrate the untold possibilities that can be made use of through the same means.

The clay I use is an Italian modeling clay, the same as sculp-

tors use, and can be used over and over again. I should mention this particular clay is mixed with vaselin and after repeated usage may become slightly dry. If this occurs, the remedy is more vaselin and we have it as good as ever. The colored clay is Plasticine and may be obtained in numerous colors from any art store. I shall first demonstrate the nasal septum, next the lateral nasal wall and the ethmoid capsule.

DISCUSSION.

DR. JOSEPH C. BECK, Chicago, Illinois: I consider this one of the best things I have obtained at this meeting. I also wish to bring up the name of one of our old Academy members, Dr. Wm. Ballenger of Chicago, who used modeling clay in demonstrating to students, and he could teach all we wish to teach by this method. I have never adopted it, because he never showed it to me as plainly as Dr. Ridpath has today. I use a book of large sheets, as big as that blackboard, made up of ordinary wall paper, but it is unwieldy to go back in this way to our illustrations of years ago. These clay models are not preserved, but there is no comparison between the value of Dr. Ridpath's and mine. I think one of the important things is the use of different colors to bring out the different divisions. I am very thankful to Dr. Ridpath for his demonstration.

DR. RALPH A. FENTON, Portland, Oregon: This splendid demonstration of Dr. Ridpath's is the first I have seen in this way, although it has been used in osteology courses for some time. I understand Dr. Ridpath uses several different colors of clay in his own clinic, and it is to my mind one of the most brilliant teaching methods that has come out in recent years.

DR. ROBERT W. BLEDSOE, Covington, Kentucky: I would like to ask the Doctor, when closing the discussion, to repeat his formula for mixing this clay.

DR. ROBERT F. RIDPATH, Philadelphia, Pa. (closing): I thought I had brought the various colored clays which I use with me, but when I opened my grip I found that I only had the gray and the red. The time at my disposal would have allowed me only to show that with the red clay you can illustrate circulation in any portion by simply rolling and running it anywhere you wish. You can use the white clay to demonstrate the nervous system and the blue the venous system. The clay can be procured in any color. The colored clay is known as "Plasticine." It is very cheap, costing only a few cents a pound, and can be procured at the five and ten cent stores. The gray is known as Italian clay No. 2, and it is obtained at the art stores. It is already mixed with vaselin. If it should become dry as time goes on, all you have to do is to mix a little more vaselin with it and it lasts indefinitely.

HERPES ZOSTER OTICUS.

J. A. WATSON, M.D., F.A.C.S.

MINNEAPOLIS, MINN.

CASE 1. E. A. B., male, age 42. I saw this patient at his home on Nov. 5, 1918. He had been having pain in the right ear since the afternoon of Nov. 3rd. The pain was deep and boring in character. He was apparently a healthy and vigorous man. The personal and family histories were negative. He had no recent acute disease, no cold, no sore throat, no cough, no influenza. There were no evident focal infections.

The drum membrane was decidedly reddened, though without any definite bulging. Nevertheless, I was considering the advisability of suggesting a paracentesis, as he lived in a rather inaccessible district on the very outskirts of the city, and I feared that he was in for an uncomfortable night, when he informed me that his face on that side felt stiff and numb. It was at once evident that there was a slight though definite paralysis of the right facial nerve, involving both the upper and lower groups of fibers. Further examination of the ear revealed the fact that the redness was not confined to the drum membrane, but involved the whole circumference of the inner half of the canal, though the color was not so marked as on the drum itself. At a later stage, there was a definite numbness of the concha. This numbness was probably present even in the early stage of the disease, but at that time I did not search for it, never having recognized a case before and not being very familiar with the symptom complex. However, I did make an immediate diagnosis of herpes zoster oticus, in spite of the absence of any herpes, and informed the patient that a rash was almost certain to appear shortly. It was not, however, until three days later, viz., on Nov. 8th, that my forecast was justified by the appearance of numerous vesicles, closely covering the concha and the canal wall, as far as it was visible. The latter, however, was now so swollen that it was impossible to examine its deeper portion, and I am still uncertain whether the drum itself was involved in the vesicular outbreak. There are a few small scars on the concha, but none that I can determine on the drum or in the canal. There were no throat or intraoral symptoms of any kind.

The pain and rash subsided gradually. The paralysis did not entirely disappear for about a month. The hearing was left

unimpaired, though during the acute manifestations there was a very considerable degree of deafness of the obstructive type.

CASE 2. O. H. T., male, age 42, consulted me in the office, Dec. 6, 1920. His left ear had begun to pain on Nov. 25th, following a ride in the cold. There had been more or less pain since, though not so severe as at first. There had been no discharge at any time. In the beginning, the pain was accompanied by vomiting, dizziness, and deafness, and the dizziness and deafness still persisted. The personal and family histories were negative. He had always been in good health, and had had no cold or influenzal attack prior to the onset of the trouble. Unlike the former case, deafness was practically complete in the affected ear. Weber lateralized to the sound side. The function of the vestibular nerve was also completely interrupted, as evidenced by the caloric and rotary tests. There was numbness of the skin of the concha and a definite redness of the drum membrane and deeper portion of the canal, but no vesicles at any time, unless there were vesicles during the eleven days between the onset of the disease and the first time I saw him, which is unlikely, as there were no scars or other traces of them. There were no throat or intraoral symptoms. There was a very definite paralysis of the left facial nerve, involving especially the superior fibers. In spite of the absence of a rash, I diagnosed the condition as herpes zoster oticus, or rather as ganglionitis, affecting the geniculate and auditory ganglia, since the former term under such a condition is of course a very paradoxical one.

This patient improved rapidly, so that in a month the symptoms had disappeared. But in six weeks from the time I first saw him, he died from cerebrospinal meningitis, which developed suddenly after exposure to cold at a funeral which he attended. I have been unable to obtain any details of his last illness from the doctors who attended him at his home in the country. They have no notes, but the diagnosis of meningitis was confirmed by one of our leading neurologists who saw him in consultation, and who did a spinal puncture, but is unable to remember the bacteriologic aspects of the case and has mislaid his records. It is unfortunate that there was no autopsy, as the opportunity would have been a rare one and might have revealed an involvement of several ganglia, in accordance with Hunt's conclusions, as well as determining any connection between the ganglionitis and his final fatal illness. I did not, however, learn of that illness and death until long afterwards.

Since Hunt's original articles of about fifteen years ago, a considerable number of cases have been reported showing ap-

parent involvement of the various cephalic ganglia, now of one, again of another, and yet again of more than one or even several of those ganglia, by the supposedly specific inflammation which is believed to be the cause of the herpetic eruption and other clinical symptoms. Some of the reported cases were evidently not cases of true herpes zoster, but rather a vesiculation from other causes, the reporters of these cases having failed to recognize the specific and peculiar manifestations of true herpes zoster. The fairly known and established facts regarding the disease are at present these: The essential pathology is an inflammatory—probably usually hemorrhagic—lesion of some or all of those particular cephalic ganglia which receive their innervation from the external ear or its immediate neighborhood. These ganglia are as follows: 1. The geniculate, which receives sensory fibers from the concha, a portion of the antihelix, the fossa of the antihelix, the antitragus, the incisura intertragica, a portion of the tympanic membrane, external auditory canal and meatus. 2. The ganglia of the glossopharyngeal and vagus nerves, whose auricular fibers arise from the posterior portion of the tympanic membrane and auditory canal, and part of the posterior surface of the auricle and the immediately adjoining mastoid region. 3. The Gasserian ganglion, which, so far as the ear is concerned, receives fibers from the tragus and the crura of the antihelix. To these should be added the auditory ganglia, viz., the ganglia of Scarpa and the cochlear ganglia, the distribution of whose fibers is of course entirely intralabyrinthine, rendering it impossible consequently for any lesion confined to these ganglia to exhibit herpes as one of its manifestations.

These cephalic ganglia are perfectly homologous, embryologically and morphologically, with the posterior spinal ganglia, with the exception that most of the cells of the auditory ganglia (ganglia of Scarpa and cochlear ganglia) are bipolar. It has been demonstrated however that even those ganglia contain some unipolar cells. While of course there has been little opportunity for pathologic examination of any of these ganglia following the manifestations of zona, enough has been done to demonstrate fairly conclusively that all the facts established by Head and Campbell more than twenty years ago concerning the pathology of the spinal ganglia in herpes zoster apply equally to the cephalic ganglia. Hunt was the first to assert or even suggest that herpes zoster affecting the ear was a distinct clinical entity, dependent on an inflammatory lesion of one or more of these ganglia, most commonly of the geniculate ganglion, though any of the other ganglia might be primarily or secondarily involved.

He did not rest satisfied with assertion, but demonstrated it so conclusively and scientifically, that the disease is often quite appropriately called Hunt's disease. He showed that these cephalic ganglia, with the second and third cervical ganglia, were links in an anatomic chain of ganglia receiving fibers from adjoining zones, which together cover the side of the face, head, and neck, including the auricle, external auditory canal, and tympanic membrane. The particular area involved by the herpetic eruption is of course dependent on the particular ganglion or ganglia involved.

Hunt divided all cases of auricular zona into three clinical groups, which I will mention, with comments of my own, as follows:

1. Cases of auricular herpes without any auditory symptoms and without facial palsy. These cases are of a mild type comparatively. The patient usually suffers for a few days from more or less prodromal pain in the affected ear, of a sharp, darting kind. There may be a slight fever. The characteristic rash appears in three or four days, and is nearly always confined to the geniculate zone and often to a part of that zone only, as e. g., to the concha alone, or to the auditory canal, or to the drum membrane, as in a case reported by Dabney, though the drum membrane is indeed affected only comparatively rarely. Dabney's case however does not belong in this group, as it was of a severe type and exhibited facial palsy and other symptoms which place it in another group.

2. The symptoms are the same as in the first group but with the addition of facial paralysis on the affected side. The paralysis is peripheral in type. Hunt says that its time of onset varies, commencing sometimes simultaneously with the herpes and at other times being delayed a few days or a week. I believe however that it varies more widely than this. In one of my cases it commenced several days before the appearance of the herpes.

When we consider the anatomic relationship of the facial nerve and the geniculate ganglion, it is at once evident that there need be no actual extension of the inflammatory process to the nerve fibers in order to produce paralysis. The ganglion is situated not in the fundus of the auditory canal but actually within the opening of the Fallopian aqueduct, a very small canal with unyielding bony walls, in which consequently any marked swelling of the ganglion is almost bound to compress the nerve sufficiently to interfere with its function. As a matter of fact, the paralysis in these cases usually clears up much more quickly than

that produced by a neuritis or a definite trauma, though there are a few reported cases in which it persisted for a long time, and one at least in which the recovery was never complete. This seems the proper place to draw attention to the fact that the intra-fallopian position of the ganglion, which is responsible for the very common involvement of the facial nerve, at the same time must render both branches of the auditory nerve almost immune from pressure injury. The crista falciformis, which crosses the fundus of the auditory canal horizontally between the entrances of the facial and auditory nerves into their final bony canals, below the former and above the latter, is a further protection to the auditory nerve, as the ganglion would have to swell not only actually out of the entrance of the Fallopian aqueduct, but also as it were over the edge of the crista falciformis, when that structure is actually present, before it could bring pressure to bear upon the nerve. Consequently some other explanation is necessary to account for the auditory symptoms, in cases at least where there is a normally situated ganglion, and especially where there is a well marked crista falciformis. This brings us to speak of Hunt's third clinical group, viz.:

3. Cases accompanied by facial paralysis and auditory symptoms. In his earliest communication, Hunt ascribed the auditory symptoms to the proximity of the terminal divisions of the auditory nerve. Later, however, he was evidently inclined to believe that they were due to the involvement of Scarpa's and the cochlear ganglia, not on account of their proximity to the geniculate, as indeed they are entirely separated from the latter by dense bony structures, but because these ganglia, of the same genetic origin as the other cephalic ganglia, are equally open to the selective power of the specific poison of the disease. This I believe to be the true explanation. Certainly, in some of the reported cases, these ganglia appear to have been the first attacked. My own second case, which I have no hesitation in reporting under this category, was ushered in by sudden dizziness, vomiting, and deafness. If the disease had commenced in the geniculate or any of the other cephalic ganglia, and secondarily affected the auditory fibers, either by pressure or by local extension of the inflammation to these fibers, such an onset would have been inconceivable. Neither does it appear possible that the auditory ganglia could have been only secondarily invaded. On the other hand, if the disease really did primarily involve the auditory ganglia, it would be equally impossible for the facial nerve to be affected either by pressure or by extension. The theory that the case was one of simple auditory neuritis, sec-

ondarily affecting the facial nerve by pressure or by extension is also not tenable, for it will not account for the distinct redness of the canal and tympanic membrane, or for the numbness of the geniculate skin area. The conclusion that the case was one of primary involvement of the auditory ganglia seems justified. The geniculate ganglion was probably involved only secondarily, causing sufficient swelling of that structure to produce the facial paralysis. Hunt, if he accepted the diagnosis in this case, would probably have explained the lack of definite vesiculation by the theory that the geniculate ganglion, being involved only secondarily, was involved only in a comparatively slight degree, since he dwelt at some length on this question of mild secondary involvement without vesicular outbreak in the skin area of the secondarily involved ganglion, though with a very definite outbreak often in that of the ganglion primarily attacked. This however can hardly be the true explanation of the absence of a definite zona, since cases have been reported in which there was an evident secondary involvement of one or another ganglia, which were certainly by no means mild and yet without any definite zona of the secondary ganglionic area. It is more than probable that there are just as many cases of primary ganglionic involvement without zona, cases in which a diagnosis is usually not made at all. There are cases reported in which the vesiculation appeared only late in the course of the disease, and then, as it were, with difficulty, and sometimes only in slight degree. In Dabney's case, e. g., the disease commenced on Jan. 30th, and not until March 6th did any vesicles appear and then only one, on the tympanic membrane. A second vesicle appeared in the same region as late as Apr. 13th. It may, however, be objected that this case was one of secondary otitic involvement, since the patient had suffered from an attack of facial herpes zoster some months previously. Be that as it may, the secondary involvement, if it was such, can certainly not be called mild, since all the symptoms except the vesiculation were acute and severe. I am indeed of the opinion, that the herpetic outbreak which has given to this disease the name by which it is commonly known, is not an essential feature of the disease at all.

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DISCUSSION.

DR. JOSEPH C. BECK, Chicago, Illinois: This Academy is to be congratulated upon putting on record four cases by four different men of this comparatively rare disease. Dr. Vail, twenty years ago, reported a case of herpes zoster oticus without any reference to any nerve disease or labyrinth connection, because we did not know anything about that subject at that time. Dr. Brown of Colorado, the essayists of to-day, and I have reported these four cases. Dr. Watson did not mention anything in the way of treatment, and he might in closing tell us what he did after his patient recovered.

I will confine my remarks to my own case. A surgical operation on the tonsil brought about rapid improvement in the labyrinth, as well as the facial paralysis. The patient was fifty-two years old and a good drinker of wine, who came in complaining that he had a pain in his ear. I could find nothing the matter and sent him to a dentist, whose examination was negative. Four hours later, he telephoned me and said that while he was drinking some water it trickled down one side of his mouth. He kept after me and I found a fullfledged facial paralysis, but found nothing in the ear until two days later, when I was called to see him in his home. He then had intense pain in the ear and about the face, and I noticed three tiny specks on the concha. A few hours later, I was called again and found it necessary to give

morphin to relieve the intense pain. He then had a spontaneous nystagmus and vesiculation. I did not understand the nystagmus, and then studied up J. Ramsey Hunt. The next morning, the patient was completely deaf, and the intense edema was increasing. I went into the history, and recalled that I had opened four peritonsillar abscesses for this man in the period during which I had known him. He had just returned from a hunting trip, and while he was away he had had another of these peritonsillar abscesses. There was still evidence of infection. I opened this rather widely, and the symptoms very rapidly improved so far as the labyrinth was concerned, but the facial paralysis in that portion of the nerve which controls the palpebralis, is still present. The lid does not close. The eliminative treatment and the search for a focus of infection should of course be carried out. I think the term of ganglionitis is a good one. We know this is not inflammation. There was no temperature in my case, and no change in the blood, and the earliest possible location of the septic focus is all important. We must know that this is a congestion of those parts, and the sooner we can get rid of the infection the better it will be.

DR. J. A. WATSON, Minneapolis, Minn. (closing): I have nothing further to add except to answer the query regarding treatment. I searched carefully in both cases for any focus of infection but could find none, although I always believed, as Dr. Beck says, that it did come from some such focus. I think we should always search for it and eliminate it when found. My treatment consisted of rest in bed and elimination. Further than that I did not think there was anything to do.

POLLEN AND HAY FEVER—A REGIONAL PROBLEM

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COLORADO SPRINGS, COLO.

The first completely successful treatment of hay fever was climatic. It has long been known that many of the victims of this disorder could escape their annual summer attack by a temporary or permanent change of residence, and on this account hay fever resorts have become popularly known both in the United States and Europe. In most cases, it will be found that such a center draws its clientele from regions entirely different in physical and climatic character from those at the resort. People from the open country go to the great woods; those from the interior to the seaside; from the lowlands to the mountains, and from the mountains to the sea level; in other words, they change definitely their botanic environment.

It does not require much consideration of this subject to see that we must have a sound botanic foundation for our pollen extract therapy. Plants cause hay fever only as an incident in the distribution of their pollen. The latter consists of rounded dust like particles, each of which contains a sperm destined to fuse with the egg of the ovule, to form the embryo found in each mature seed. In the bright colored flowers of the field and garden, the stamens containing the pollen and the ovary with its ovules or young seeds are located in the same flower, and the pollen is transferred from one to the other by means of insects in search of honey. In the case of most of our common trees, the grasses and the so-called "weeds," the pollen is produced in one flower and the ovary in another, often on different plants. With all such, the pollen must be carried by the wind, and it is these wind borne pollens that enter the nose and give rise to hay fever. While the pollen of insect pollinated flowers may cause hay fever, it is too heavy to be readily carried by the wind, and hence few individuals become sensitized, and symptoms can occur only when the pollen is actually snuffed into the nostrils. Hay fever supposed to be due to goldenrod, sneezeweed (*Helenium*) and other bright colored flowers, is in reality produced by other pollens. As the number of pollen grains carried by the wind decreases with the cube of the distance, the abundance of pollen diminishes very rapidly with the distance from the point of origin. Windy days carry the most pollen, though striking differences

even then occur in distribution; for example, the windward side of a small public building yielded more than twenty times as many pollen grains as the leeward.

The first step in the application of these botanic principles to the hay fever problem requires a careful survey of the region in which the hay fever to be treated occurs. Plants must be studied as to their species, occurrence and the time of flowering. While the great orders that contain practically all of the plants to be considered, namely, the grasses, composites, sunflowers, buckwheat and walnuts have representatives throughout the country, the actual species differ greatly between the eastern, middle and western states. Thus, in the Rocky Mountain region and the Mississippi Valley, there is the general group composed largely of weeds introduced from Europe, grasses and trees, which are almost identical for both east and west. The second group consists of similar weeds or escapes from cultivation, such as Russian Thistle, Lambsquarter and probably Summer Cypress, which seem to exhibit increased virulence in the Rocky Mountains. This we know to be particularly true of Lambsquarter. The third group contains such native species as the Sageworts. The latter have been on the ground for the longest time, and are being slowly displaced by cultivation and the advent of eastern weeds. They are probably to be regarded as responsible for most of the hay fever actually contracted in the locality of the writer. While it is desirable to know the wind pollinated plants of a state, such knowledge has little practical value until it is translated into terms of local occurrence and abundance. In Colorado for example, Ragweed is an important plant in and about the cities of the eastern slope, while it is unimportant or lacking on the western. The Sageworts are common on the plains at the eastern foot of the mountains and the Sagebrushes rare, while in the western half of the state Sagebrush and Saltbush are all-important, and the Sageworts of little significance. Similar differences occur with respect to altitude; Ragweeds, Russian Thistle and other introduced weeds drop out rapidly between 6,000 and 7,000 feet, while the Sageworts ascend to 9,000 and the Sagebrush even higher, as does Timothy. There are also striking contrasts in local distribution. Kochia, (the Summer Cypress) often grown in gardens, has escaped abundantly at Pueblo and is an important cause of hay fever, while in Colorado Springs, less than fifty miles north, it is so far known in but one garden, from which it has escaped into the adjacent railroad right of way.

Diagnosis. The usual method of arriving at a decision as to the offending pollen in hay fever has been to test the skin reaction of the patient with all the pollens available. If all reactions were absolutely conclusive, and if the list of pollens used was complete, the results might be satisfactory even if laborious, but it must be remembered that just because a patient gives a skin reaction to a pollen or pollens, it does not necessarily follow that that particular pollen is responsible for the hay fever. The method we have followed we believe to be more rational and scientific. It involves:

(1st) A thorough pollen survey of the whole region, supplemented if possible, by one of the patient's immediate environment.

(2nd) An accurate determination by the pollen plate method of the specific kind and relative amount of pollens around the patient's home, as soon as possible after the onset of symptoms.

(3rd) A detailed history of the patient's case.

(4th) Appropriate skin tests as indicated by the previous study.

Pollen Survey. Our survey consists of determining the date of first pollination, when the pollen becomes abundant, the date of the last pollination, and the habitat or locality of the composite, grass or tree. This has been carefully recorded in our locality for the past two years and the difference in seasons noted. It can be readily seen that this must be done by one thoroughly familiar with every species and capable of making an accurate determination. The physician himself is hardly competent to do this, so the services of a trained botanist should be sought. The best results can be obtained only by close cooperation between the physician and the botanist. It is inadvisable to rely on blanket surveys which cover too large a territory, and which attempt to give lists for each state. A survey should be made for each locality. Such surveys have been made by Prof. H. M. Hall and Dr. Grant Selfridge in California.

Pollen Plate Method. If the patient has been tested pre-seasonally and immunizing injections given, and later develops hay fever¹, Petri dishes or glass slides smeared lightly with glue or mineral oil are placed on his sleeping porch or window-sill. The wind borne pollens will adhere to this, and can then be identified microscopically. After noting the plants in flower in the neighborhood, the identification of the various pollens becomes a simple matter; no more difficult than the differentiation of common bacteria.

History. It is not so important to know how long a patient has had hay fever as it is to learn when and where he first developed it; whether he has gone through a season without an attack, and if so, where; the date when the attacks appear and their duration. The date and duration of the attack can then be compared with the pollen survey of his own region, and the list of plants likely to be responsible narrowed to a comparatively small number.

Skin Test. In our tests we have used both the scratch method and the intradermal. We find that the latter gives more sensitive indications, and that some individuals who give a very slight or negative reaction to the scratch test give as much as a three plus reaction with the intradermal. This bears out the findings of Brown². In using the dried pollen, there is greater likelihood of contamination either by currents of air or instrumentation, and this possible source of error I do not believe has been given sufficient attention. Both care and experience are essential in the clinical interpretation of skin reactions. The conclusions of Larson, Paddock and Alexander³ in this connection are as follows: "The skin reactions are influenced by the following factors": (A) The preparation used. (B) The method of application. (C) The length of scratch made. (D) The site of injection. (E) The degree of cellular sensitivity. (F) The amount of protein brought in contact with the cells and the amount of solution injected. And to these the writer would add, the time the patient is tested, whether preseasonal or after the onset of symptoms. I find the reactions are more marked after the attack begins. Severe systemic reactions from skin tests will occur, but these can be greatly reduced by starting with weaker solutions and increasing the strength.

Collection and Preparation of Pollen. The collection of pollen is a task that demands both knowledge and care. The species must be accurately known and its flowering period must be followed in some detail. Securing an adequate amount of pollen is a time consuming task, and for this reason it can be done most economically during the period of maximum flowering. It is usually desirable to take entire plants or large portions of the flowering part, as under favorable conditions anthers may mature and shed pollen for several days. The actual collecting of the pollen itself is best done in a room free from dust and movements of air. The pollen is allowed to drop on sheets of white paper and it is then passed through a sieve of bolting cloth to remove flower particles, dust, etc. Contamination is avoided by

placing the various clusters at such a distance from each other that movement about the room will not cause admixture with another pollen. The latter is fatal to trustworthy results, just as the practice of grinding up flowers or inflorescences make it impossible to properly standardize the extracts used, or to be certain of the role played by the chemical constituents and the flower parts or leaves. Before being bottled, pollen must be thoroughly air dried, or better still, dried in a water bath at low temperature to keep out moulds and bacteria. The plants should be collected away from traveled roads and dusty places to secure them as clean as possible. It is also best to anticipate the pollinating time by several days, in order to insure pollination in the collecting rooms. It is a wise precaution to keep voucher specimens of all material collected.

Treatment. We have endeavored whenever possible to carry out the preseasonal diagnosis and immunization treatment; however, a great many cases fail to present themselves until the attack is on. The average hay fever patient is a confirmed optimist prior to his attack, and he is invariably sure that the coming season will find him free from symptoms. Our experience in the pollen extract therapy during the attack is growing more encouraging each year, and this I believe is due to more thorough study of the individual case and the accumulating experience from our local plant and pollen surveys. The difficulties in diagnosis are becoming less as we acquire more knowledge of the common offenders in our local fields. Dosage requires closest attention and must be regulated differently for almost every patient, this being especially true in seasonal treatment. The patient should be under direct supervision from the time he starts his treatment until his season is over if the best results are to be obtained.⁴ Stronger solutions are more effective, but the dosage must be guarded to prevent reactions. During the season, relief is not infrequently noted following the skin tests. Just as patients react differently to the preliminary tests, so do their needs and reactions vary in treatment; fixed rules for dosage cannot be laid down.

Preparation of Pollen Extracts. In preparing extracts, we have followed the method worked out by I. Chandler Walker⁵, using a 12% alcoholic normal salt solution. A stock extract of a 1 to 100 solution is made first, and from this other dilutions are made as required, up to 1 to 50,000.

Case Reports. I would like to report three cases to illustrate the necessity of local plant surveys and the use of pollens gathered

in the region in which the hay fever begins. (1) A physician who had suffered from hay fever for a number of years was tested by a specialist of large experience with negative results; some thirty-five different pollens were used, but these were gathered from a section of the country about a thousand miles from the patient's home. Hay fever developed at the usual time. A survey was made of his neighborhood and Russian Thistle and Lambsquarter were found in abundance in a nearby lot; pollen plates set on a window ledge of his bedroom showed granules from Pine, Lambsquarter and Russian Thistle. It was immediately evident that Pine was not the offender, and a test of the other two pollens showed a four plus reaction to Lambsquarter and a negative reaction to Russian Thistle. Immediate benefit was obtained from the appropriate extract, and the physician has been free from irritation for the past two seasons.

(2) A woman who has resided in Colorado Springs for twenty years and has had hay fever each season, was given a full course of immunizing injections in the East. She had been found to react to Ragweed. The following season, in Colorado, her hay fever showed no improvement and she was very much discouraged. The two following seasons were spent in the vicinity of Philadelphia at a country home, with complete relief from symptoms; the third summer was spent in Colorado Springs, and hay fever reappeared as usual; the date of her first attack coincided with the appearance of Sage pollen, and she was accordingly tested with this pollen; the reaction was unusually severe, but she obtained complete relief from symptoms for two days following the test. The use of weak solutions every few days kept her practically free from irritation for the balance of the season.

(3) A business man sensitive to Ragweed was thoroughly immunized and had no hay fever for two seasons in Colorado Springs. His business then called him to Pueblo only forty-five miles away; he soon telephoned that his hay fever had returned in a severe form and that he was coming to see me; he was tested to every pollen that we possessed, with negative results; even the Ragweeds which had previously caused his hay fever, gave no reaction. Our botanist was sent to his new surroundings to make a survey and found an unknown weed in great abundance on the vacant lots surrounding his home. Pollen gathered from this plant gave a strong reaction, and it was not until authoritative opinion was obtained, that this plant was found to be the ordinary Summer Cypress (*Kochia*) often grown in gardens, which in this instance had escaped and was growing wild.

SUMMARY.

(1) Hay fever is caused almost exclusively by plants whose pollen is sufficiently light and abundant to be distributed widely through the air.

(2) Many of these plants are limited to certain sections of the country, or are unevenly distributed, and in consequence, the etiology of hay fever becomes largely a regional problem.

(3) Much of the failure in pollen extract therapy is due to an insufficient knowledge of the botany of plants with wind borne pollen.

(4) If the best results are to be obtained, a survey of such plants should be made by competent authorities for each locality, to determine the species present, their relative abundance and pollenating time.

(5) The individual case should be treated with a full knowledge of the significant plant life in the region in which his hay fever develops.

(6) Diagnosis is simplified by a thorough study of the history of each case in connection with an adequate local pollen survey, including pollen plating.

(7) Treatment with pollens gathered in the region in which the attacks occur is always advisable and often essential.

(8) Treatment during attacks presents many difficulties, but promises increasingly good results.

In closing, the writer wishes to express his sincere gratitude to Robert A. Cooke of New York for his valuable help and ever willing suggestions; to I. Chandler Walker, for giving to us the minutest detail of his years of laboratory and clinical experience; to Grant Selfridge of San Francisco, who first stimulated the interest of the writer, and showed him the needs of a pollen survey; to Drs. Fredric E. Clements and Edith S. Clements of the Carnegie Institution, for their expert identification of pollens and plants, and for illustrations and charts; and to Prof. Ralph Gilmore of Colorado College for his untiring work in pollen collecting.

Table 1. Season 1922.

24%	Reacted to Spring Pollens
19%	“ “ <i>Populus deltoides</i> (Cottonwood)
35%	“ “ Russian Thistle
35%	“ “ <i>Chenopodium</i> (Lambsquarter)
25%	“ alike to Russian Thistle and Lambsquarter
23%	“ to <i>Kochia</i> (Summer Cypress)
5%	“ “ <i>Zea Mais</i> (Corn)
Less than 5% Reacted to various grasses.	
33%	Plus Reacted to <i>Ambrosia Trifida</i> (Giant Ragweed)
44%	Reacted to <i>Ambrosia Artemesifolia</i> (Common Ragweed)
27%	Reacted to <i>Iva Xanthifolia</i> (Marsh Elder, Prairie Ragweed)
66%	Reacted to <i>Artemesia Campestris</i> . Field Sage Wort.
Note—Percentages based on total number of cases tested.	

<i>Distichlis spicata</i>	6/22	6/15	6/20	7/15	Cultivated	1cc
<i>Stipa viridula</i>	6/22	6/15	8/10	Damp places (not common).....	1cc
<i>Amaranthus retroflexus</i>	6/25	6/25	7/1	7/15	Garden weed	2cc
<i>Plantago major</i>	6/29	6/25	7/1	Frost	Lawn weed	1/2cc
<i>Phleum pratense</i>	6/29	6/20	7/15	8/15	Alkaline Soil (not common).....	1cc
<i>Boottlova oligostachya</i>	6/30	7/5	8/20	8/10	9/10	Wild grass Mesa (not common).....	2cc
<i>Agrostis alba</i>	6/30	7/5	Frost	7/5	8/10	Waste places (abundant).....	1cc
<i>Ambrosia artemesifolia</i>	7/1	7/5	7/5	8/10	Cultivated	2cc
<i>Ambrosia trifida</i>	7/7	7/14	Frost	7/5	8/10	Waste places (abundant).....	25cc
<i>Parthenocissus quinquefolia</i>	7/8	7/15	Frost	7/5	7/25	Vine on building	1/2cc
<i>Salsola kali</i>	7/8	7/10	8/10	Waste places (abundant).....	10cc
<i>Calamagrostis longifolia</i>	8/1	9/1	Frost Wild grass.....	1/2cc
<i>Franseria acanthioides</i>	7/20	8/1	Frost Moist waste places.....	5cc
<i>Xanthium canadense</i>	7/25	8/5	7/20	9/1	Cultivated	20cc
<i>Zea mais</i>	7/25	8/5	7/15	9/1	Cultivated	10cc
<i>Zea mais</i>	7/25	8/5	Frost	7/15	9/1	Roadsides, not com., 1921, (abundant).....	20cc
<i>Artemisia canadensis</i>	7/25	8/5	8/1	8/25	Waste fields (abundant).....	20cc
<i>Andropogon forcatius</i>	8/1	8/25	Wild Grass	1cc
<i>Homulus lypolus</i>	8/1	8/25	Underbrush Waste Places.....	10cc
<i>Sporobolus cryptandrus</i>	8/1	8/25	Common waste dry fields.....	1/2cc
<i>Iva xanthifolia</i>	8/4	8/20	Frost	8/15	8/25	Waste places (abundant).....	10cc
<i>Eurotia lanata</i>	8/10	8/10	9/10	Sage-like plant, not abundant.....	10cc
<i>Kochia</i>	8/10	Frost	8/10	9/10	Escaped cultivation (common).....	10cc
<i>Artemesia dracunculoides</i>	8/10	8/20	9/10	Frost Not common as canadensis.....	5cc
<i>Artemesia grigida</i>	8/10	8/20	Frost	8/25	9/1	Frost Very common, mesa, foothills.....	10cc
<i>Artemesia gnaphalodes</i>	8/25	9/1	Frost Not common	1cc
<i>Artemesia tridentata</i>	Frost 1 specimen in Colo. Springs.....	1cc
<i>Thalesperma trifidam</i>	6/20	6/15	7/1	7/26	Waste places—City.....	1cc
<i>Medicago</i>	6/15	Frost	7/1	7/26	Cultivated dust and pollen.....	10cc
<i>Melilotus alba</i>	7/1	7/26	Cultivated and wild dust and pollen.....	10cc
<i>Trifolium repens</i>	7/26	Frost Lawns, dust and pollen.....	10cc
<i>Solidago canadensis</i>	7/26	Frost Wild.....	1cc

OF DOUBTFUL VALUE.

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<i>Stipa viridula</i>	6/22	6/15	8/10	Damp places (not common).....	1cc
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<i>Plantago major</i>	6/29	6/25	7/1	Frost	Lawn weed	1/2cc
<i>Phleum pratense</i>	6/29	6/20	7/15	8/15	Alkaline Soil (not common).....	1cc
<i>Boottlova oligostachya</i>	6/30	7/5	8/20	8/10	9/10	Wild grass Mesa (not common).....	2cc
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<i>Artemesia gnaphalodes</i>	8/25	9/1	Frost Not common	1cc
<i>Artemesia tridentata</i>	Frost 1 specimen in Colo. Springs.....	1cc

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DISCUSSION.

DR. JOHN B. McMURRAY, Washington, Pa.: I am sure we are all indebted to Dr. Mullin for bringing before us his work. We all have such cases in all sections of the country. We have some successes, but a much larger percentage of failures, and we have often wondered why we succeeded in some cases and failed in others. I am sure Dr. Mullin has brought to our attention some of the causes for our failures. Dr. Mullin tells us a careful survey of the pollens of the neighborhood in which a patient lives is necessary, and if we use the pollens found we will have good results. I think he has convinced you that there is an advantage in his method of procedure. In the East, I believe no one has ever considered Lambsquarter as being a source of hay fever, and yet it grows in abundance in Pennsylvania, as high as two feet in Washington County, and yet it has never been suspected. On the other hand, we have corn suspected by everyone, and yet Dr. Mullins has found that only 5 percent react to corn. This year, strange as it may seem, I had one patient who reacted very strongly to corn alone. This year is the first that this patient has ever been able to go into the garden to gather roasting ears. I can see that, as the Doctor tells us, it is a very heavy pollen. The patient would not get it in the air any distance from the field.

I can see that we are going to do a great deal of hard work if we follow Dr. Mullin. We are not all so well located that we can have the services of a botanist, and yet if we make a survey in each community and find out what is causing the trouble in our patients, it will be of much benefit to all of us. It might be done in a group way, but some way or other we must get over this difficulty.

We do not know much about sensitization, and that was not mentioned by the essayist. He very wisely omitted it. There is another statement which we should consider. He made the statement that, in some cases, in testing for skin reaction during the season of the pollen, the patient often got relief from the symptoms. The patient was better following the small dosage used for the skin test; that surprises me. I have always hesitated to test or treat after the symptoms had developed. I think, with this understanding, we might be able to do more for the patient after the symptoms have been instituted, in the season in which they are affected.

Dr. Mullin spoke about the treatment. That is very important. I think we should not accept from the average manufacturer certain set doses for this or that condition. We should study each case carefully and find out how much of a particular pollen he can take without reaction. We must never get a reaction in the treatment of these cases, and that point the essayist has also mentioned.

We do not have in Western Pennsylvania any of the sages, but we have an abundance of ragweed, and when I go home I am going to hunt for the Lambsquarter and find out if some of the patients are susceptible to it. It is probably true that in some patients we will get results from ragweed and in others from the Lambsquarter.

DR. W. V. MULLIN, Colorado Springs, Colorado (closing): Dr. McMurray spoke of drug house extracts. Even if you are giving your patient the exact extract he is sensitive to, which is put out by the drug house, it is not as strong as the extracts made in your own laboratory. The manufacturer must standardize their's according to the "Bureau of Standards" at Washington. Dr. Van der Veer exemplifies this point very well in an excellent article in the March issue of "The Journal of Immunology", showing conclusively that better results are obtained from using stronger solutions and endeavoring to increase the dosage as high as possible, without producing undesirable reactions. I cannot explain why some patients get benefit following their tests, in placing the small amounts of the strong solution intradermally.

I shall be glad if Dr. McMurray will investigate *Chenopodium* (Lambsquarter) in his vicinity, for I should like very much to know if it is harmless in some parts of the country, and more virulent in other localities. I should never have suspected it of having been a hay fever producer, had we not set out the pollen plates on the window ledge of the physician in the case referred to in my paper. He was so bigoted because he had not found anything that he reacted to, and he had taken stock extracts, so that he would hardly submit to a test, and it was only after finding these pollen grains on the pollen plate with the microscope, getting a marked skin reaction with the test, that we were prompted to suspect it in a great many other cases.

NEW INSTRUMENTS AND APPLIANCES*

1. DRESSINGS FOR WOUNDS.

HARRY L. POLLOCK, M.D. AND FRANCIS L. LEDERER, M.D.

CHICAGO, ILL.

A suggestion in the way of open air treatment of wounds by means of a dental compound dressing was offered. These dressings were used in the treatment of mastoid wounds, external nasal wounds, eye wounds and many other areas about the

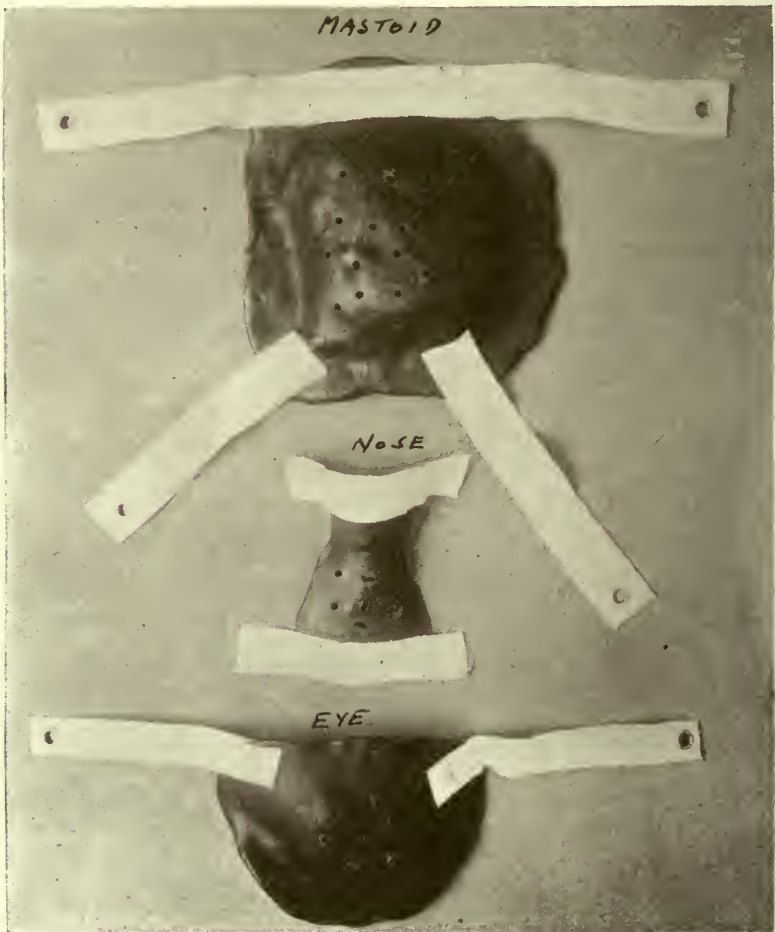


Fig. 1.

*From the Clinic of Drs. Jos. C. Beck and Harry L. Pollock, Chicago.

head and neck, where cumbersome and also air tight dressings had been used heretofore, and thereby had slower healing. These shields are easily made by immersing the dental compound in hot water and moulding it into any form desired. Perforations likewise are made in any number. These shields are made to fit each individual patient, and tapes are attached to the shields to go around the patient's head, or they are fastened by adhesive to the skin surface. (See Figs. 1, 2 and 3.)



Fig. 2.

They also presented a dressing ideally suited for tracheotomy or laryngectomy wounds. This consisted of a rubber bib or apron, with an opening or slit through which the tracheotomy tube was placed, which would protect the skin from the irritative secretions and keep the dressings from soiling so readily. The use of rubber as a material was advised because secretions were easily wiped off with a wet cloth, and also because rubber was easily sterilized. (See Figs. 4 and 5.)

Dr. Pollock also suggested that tracheotomy tubes should be made with two inner tubes and one outer tube, thus avoiding the necessity of buying two complete sets which heretofore had been necessary. Mueller & Co. have signified their willingness to cooperate in this respect.



Fig. 3.

2. ARTIFICIAL LARYNX.

HARRY L. POLLOCK, M.D. AND FRANCIS L. LEDERER, M.D.
CHICAGO, ILL.

An artificial larynx was presented, which consisted in the main of a tracheal canula and a pharyngeal canula, the latter being made of flexible rubber in order to prevent irritation of the epiglottis that results when a metallic tube, which does not give, is used. A diaphragm arrangement between the two allows for phonation. The instrument thus far is still in its experimental stage, but even so far as it has gone, it permits a laryngectomized patient's voice to carry 10 to 12 feet.

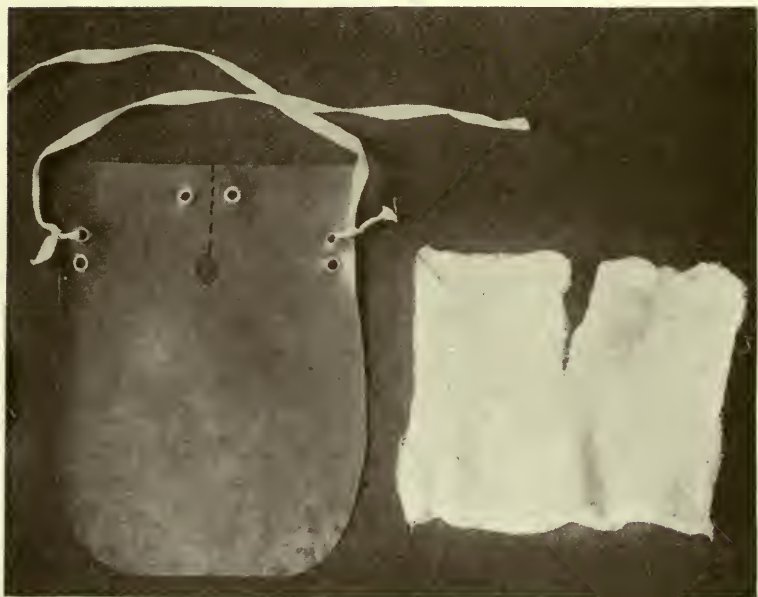


Fig. 4.



Fig. 5.

3. WALL PLATE, IRRIGATION BOTTLE AND CANULAE.

DR. JOSEPH C. BECK, M.D.

CHICAGO, ILL.

The wall plate consisted of a suction and air pressure outfit, together with a transformer and electric light attachments. This plate was a compact unit which could be used in any office. There was no noise connected with the suction or air, as the motor for the same was in the basement. V. Mueller & Company are the makers of this instrument. (See Figs. 6, 7 and 8.)



Fig. 6.

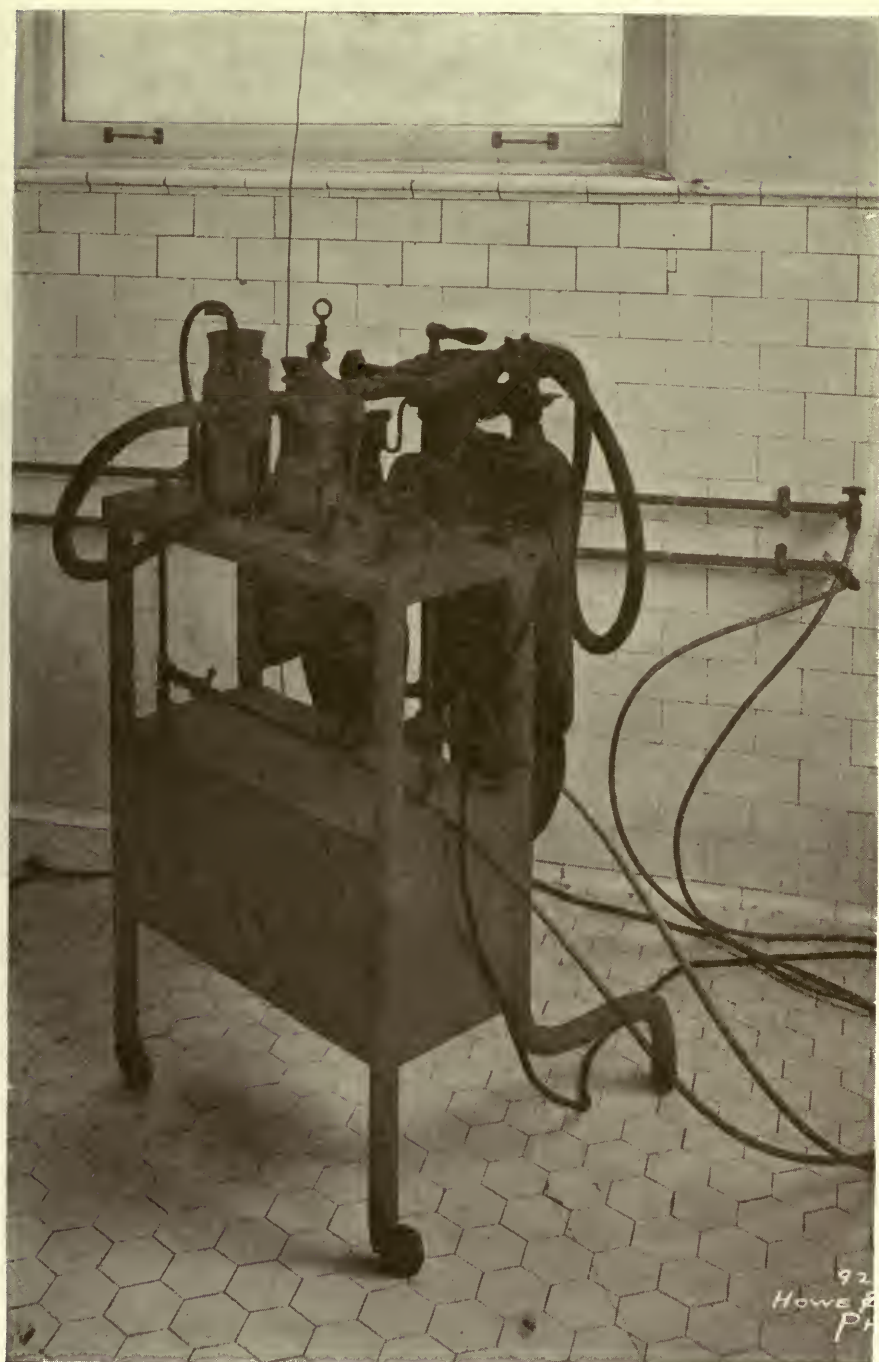


Fig. 7.

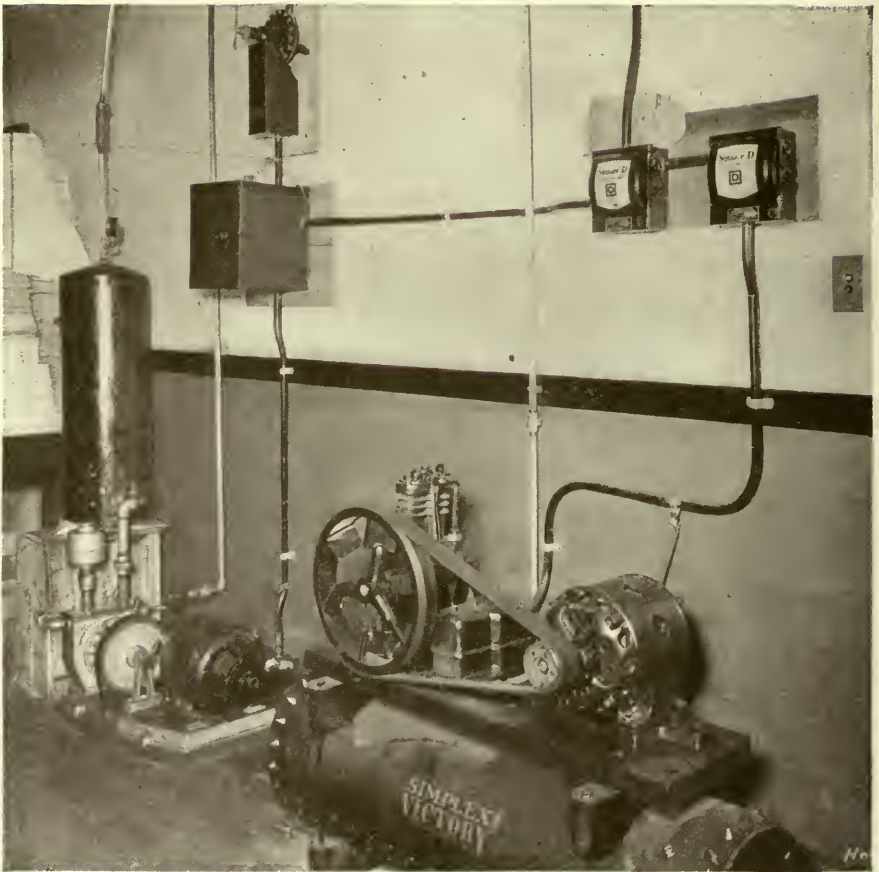


Fig. 8.

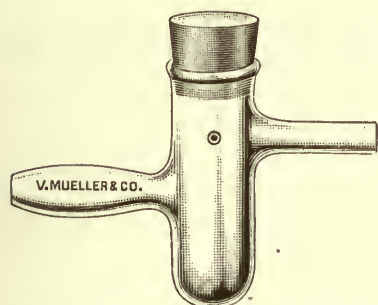
Dr. Beck also presented an irrigation thermos bottle which he had devised, the water being kept at a warm temperature during the working hours.

He also showed various types (6 in number) combination suction and irrigation metal canula, which suit any use about the head and neck. Both the irrigation bottle and the canulae are being made by Sharp & Smith. (Bottle seen in Fig. 6.)

A NEW NASAL SUCTION TIP FOR TREATMENT OF THE PARANASAL SINUSES.

HORACE NEWHART, M.D.,
MINNEAPOLIS, MINNESOTA.

The glass tip herewith presented is an improvement on the old form, which we have used for many years without knowing the name of the original designer, to whom we should like to give credit.



The chief disadvantage of the old tip was, that not infrequently during the act of aspiration, the evacuated contents of the sinuses and nasal discharge would jump across the body of the receptacle and

soil the suction tube leading to the pump—a most annoying occurrence. This disadvantage has been overcome by having the glass blower place the inflow and outflow tubes in such a position with respect to each other, as to make this passing of the discharge across the body of the receptacle impossible, thus obviating such an unpleasant occurrence.

(2) A NEW MASTOID PROBE AND INSTRUMENT FOR REMOVING FOREIGN BODIES FROM THE AURICULAR CANAL.

HORACE NEWHART, M.D.,
MINNESOTA.

An instrument which we have found most useful in otologic practice is a modification of the original Jansen mastoid probe, which seems to be but little used in this country.



We have modified it to the extent of providing it with a larger and longer handle, so marked that the surgeon always knows the position and direction of the point or blade. The point has also been somewhat dulled. The instrument is used regularly in all mastoid operations as a probe, its point being such that it is impossible to injure the dura or the lateral sinus. It is of great help in exploring in advance of removing bone. We have also found this simple instrument most useful in the removal of foreign bodies and cerumen from the auricular canal.

FOREIGN BODY INSTRUMENTS.

GEORGE W. BOOT, M.D.

CHICAGO.

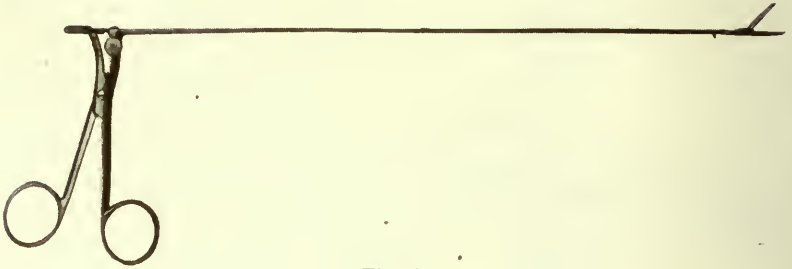


Fig. 1.



Fig. 2.

One instrument was a forcep for the extraction of peanut shells and similar foreign bodies. It is of very light construction, and small enough to pass through the smallest bronchoscopic tube. It is so made as to eliminate friction and preserve the sense of touch, which is important if fragile foreign bodies are to be removed without crushing. (Fig. 1.)

The other was an instrument for aid in the removal of staples. This instrument is a cylinder of metal, with the upper end closed obliquely and attached to a copper wire. This instrument is passed through the tube and placed over one point of the staple; the tube is then turned and the other point of the staple grasped with the forceps and the whole removed together.

TRANSACTIONS
OF THE
TWENTY-SEVENTH ANNUAL MEETING
OF THE
American
Academy of Ophthalmology
and Oto-Laryngology

NECROLOGY
MINUTES
DIRECTORY

REQUIESCANT IN PACE

Dr. K. P. Battle, Raleigh, N. C.

Dr. C. D. Conkey, Duluth, Minn.

Dr. Alanson W. Hawley, Seattle, Wash.

Dr. L. H. Landman, Cincinnati, Ohio.

Dr. H. L. Lynah, New York, N. Y.

Dr. Fontaine B. Moore, Memphis, Tenn.

Dr. J. R. Robinson, Colorado Springs, Colo.

Dr. Charles C. Stuart, Cleveland, Ohio.

Dr. C. W. Tangeman, Cincinnati, Ohio.

MINUTES

BUSINESS SESSIONS

The first Business Session was held Tuesday morning, September 19th, 1922, following the formal opening of the Academy Sessions.

It was moved by Dr. C. F. WÜRDEMANN, Seattle, Washington, that the reading of the minutes of the last meeting be dispensed with. Motion seconded and carried.

The SECRETARY then read his report, as follows:

SECRETARY'S REPORT

The membership of the Academy now totals 1,390. There occurred during the year nine deaths. Through an error in the Secretary's office, the name of Doctor Charles F. Adams of Trenton, New Jersey, appears in the list. I am happy to announce that Doctor Adams is living and well, and present at this meeting. The Secretary's humble apologies have been made to Doctor Adams for this unfortunate error in printing.

Ninety-seven names appear on the list of applicants to be acted upon at this meeting of the Academy. Others have filed applications, but too late to have their names forwarded to the members, according to the new By-Law, thirty days before the annual meeting. Their names will come up for action at the annual meeting in 1923.

In making up the program, the Committee experiences considerable difficulty in assigning discussors for papers when the essayists fail to express a preference. If Fellows who intend to come to the meeting, and who do not wish to offer a paper for the program, will signify their willingness to take part in discussions, it will greatly assist the Program Committee in assigning discussors for papers in which they are especially interested. It is an honor to appear on the Academy's program, and the opening of a discussion is quite as important as the reading of the paper. The Committee will welcome cooperation of the membership in this matter.

The attention of the membership should be directed to a clause in the By-Laws which relates to the qualifications of applicants. Only those who are engaged in the practice of Ophthalmology or Oto-Laryngology, or both, exclusively, are eligible for membership, and recommenders should have per-

sonal knowledge of the candidate, and should satisfy themselves that the candidate meets the requirements of the Academy.

Instead of the usual posting of names of candidates to be acted on at this meeting, the list has been placed in the program. If any member has definite information as to questionable qualifications or ethical standing of any candidate whose name appears on the list, such information should be given to a member of the Council or to the Secretary to present the same to the Council.

The rapid growth of the Academy to almost fourteen hundred members, makes it exceedingly important to keep correct records of the names and addresses of its members and the specialties which they practice. The Secretary will therefore welcome corrections, in writing, of any errors which have crept into our records.

So many inquiries are directed to the Secretary's office as to why a member has not received his Transactions, that it is pertinent to announce again that the Transactions are sent only to those whose dues are paid on or before March 1st following the Annual Meeting. Other reasons occasionally are responsible for failure to receive the Transactions, but failure to pay dues before March 1st is the usual cause.

Respectfully submitted,

LUTHER C. PETER,
SECRETARY.

It was moved by Dr. EMIL MAYER of New York City, that the Secretary's report be received and placed on the records. Motion seconded and carried.

TREASURER'S REPORT

Statement for year ending July 31st, 1922

Balance on hand from 1921—Cash.....	\$ 3,921.82
Liberty Bonds	27,129.71
Total	\$31,051.53
Receipts for year—from dues, etc.....	\$15,511.24
Profit from sale L. B.	1,926.69
Accrued interest on L. B.	346.50
Total	\$17,773.43
Grand total	\$48,824.96

Disbursements for year, Sundry per Statement....	\$ 7,827.25
Accrued interest on total	225.32
	<hr/>
Total	\$ 8,052.57
Balance	\$40,772.39
Cash in bank 1922 Savings	\$ 6,573.87
Commercial	620.02
	<hr/>
	\$ 7,193.89
Securities on Hand	\$33,578.50
	<hr/>
Total	\$40,772.39
4-1000 Beatrice Apartment.	
5-1000 Consolidated Stone.	
5-1000 Dominion Canada.	
5-1000 Witherbee Sherman.	
10-1000 Van Sweringen.	
5-1000 State Randolph.	

SECORD H. LARGE, Treasurer.

It was moved by Dr. EDWARD B. HECKEL of Pittsburgh, that this report be received and referred to the Auditing Committee. Motion seconded and carried.

The President appointed the following Auditing Committee:

Dr. JOSEPH C. BECK

Dr. WM. L. DAYTON.

The President then called for reports of Special Committees.

REPORT OF COMMITTEE ON INTERNATIONAL CONGRESS

I think that there has emanated so much printed matter from the joint committee of the International Congress that we may be allowed to make a verbal report.

The work has been almost finished, all except the volume of transactions, and it is before the profession to study and make yourselves familiar with what particulars you choose.

There only remains this, to report to this body, as one of the bodies that joined in planning and supervising the gathering at Washington. It should be reported that there will be something of a deficit. The transactions are larger than we expected. It will be probably the largest of any of the transactions of the International Congresses, and the cost

of printing is higher than ever before. How much of a deficit there will be, we will not know until the printing is finished. When that occurs, I believe that the subcommittee on finance will make a written report to all of the members of the Academy, and probably to all the members of the Congress, stating just what it is. That deficit will be taken care of without any trouble, because a long list of men have pledged themselves to share equally in any deficit. The more pledges we get the smaller the amount will be for each individual. I think we should consider the advantage to us of being able to attend an International Congress in this country and not having to lose a month, as well as the expenses of attending the Congress abroad, such as has always been the case before.

E. JACKSON.

It was moved by Dr. EMIL MAYER that the report be received and the Committee discharged, with the thanks of the Academy. Motion seconded and carried.

It was later moved by Dr. E. C. ELLETT of Memphis, that in view of the strenuous work of this committee for a period of two years (especially the Secretary of the Academy, who in addition to his work on the Committee acted as Secretary and Treasurer of the Congress) the members be given a rising vote of thanks for their splendid services in this connection. Motion seconded and unanimously carried.

REPORT OF THE COMMITTEE ON A NATIONAL MEDICAL RESEARCH LABORATORY

In a preliminary report of this committee last year, a brief outline was given of the efforts which had been made by the American Medical Association and its friends in Congress to secure legislation which would provide for a Department of Public Health in our Government. Such a Department would have its representative in the Cabinet, it would bring together the Public Health Service from the Department of the Treasury, much of the biologic work from the Department of Agriculture and other cognate agencies now widely separated. An important part of such a proposed department would be its Bureau of Medical Research.

The objects of that Bureau were given as primarily to prepare a bibliography, with titles and abstracts, of all important medical literature. In a word, the first and most important function of such a Bureau would be the publication of what is practically a Year Book of some or all the various departments of

medicine. Next to that, the Bureau would endeavor to encourage the study of new problems and research by individuals or groups, and perform other duties already described.

It is unnecessary to call attention again to the various obstacles to any such beneficent legislation, but thus far patent medicine manufacturers, irregular practitioners and the ignorance or carelessness of the public, with various malign influences, have defeated every effort at progress which, emanating from a Department of our Federal Government, would extend outward toward the medical professions and thence to the public.

Therefore, instead of waiting year after year until such a step in advance is made, individual practitioners or groups of specialists in their various societies must accomplish as much as they can towards fulfilling the functions which have just been indicated as those which belong to a National Bureau of Medical Research.

We have just seen that the first and most important function of such a Bureau is to collect and preserve the literature of each year, and to present it, if possible, in the form of a Year Book.

There is no question that this should be done, not only to preserve a record of facts already published, to enlighten and assist us in the routine of daily practice, but also, if we attempt to write, to save us from those imaginary discoveries, the accounts of which in the form of vain repetitions form a large part of the proceedings of most medical societies.

Efforts have already been made to satisfy the deficiencies here indicated. Thus, for General Medicine we have the invaluable *Index Medicus*; but that gives only titles without abstracts.

The plan now existent of supplying all physicians with abstracts of the current literature, moulded on the one successfully inaugurated during the War for the American Red Cross by one of this Committee, is excellent, but the list is far from complete. Many of its abstracts are necessarily made by those who work solely for pay and not by experts in love with their subjects.

We believe that this work should be done by medical men, and that they should be properly remunerated for their share of the work.

Many of the journals devoted to special branches of medicine and surgery also give reviews, titles and abstracts, but the lists as far as can be ascertained, are decidedly imperfect. It is impossible to consider in detail those records of current literature, even that which relates to otology and laryngology.

Without much question, more care has been exercised to classify, to review, to abstract and to index each year the literature of ophthalmology than of any other branch of medicine. We desire therefore to call attention to that.

From 1870, Nagel's *Jahresbericht* gave practically a complete list and digest of the literature of ophthalmology, and was each year a mine of information. The volumes are still exceedingly useful, if only to show what a large part of what now seems new is really old. But the publication was arrested by the War.

The Ophthalmic Year Book, founded a few years ago, and its successor, *Ophthalmic Literature*, is by far the best of its kind in English, though not as complete as the earlier *Jahresbericht*. But unfortunately, the able editor, Doctor Jackson, to whom we all owe so much, has decided that he must leave its management in other hands, and its destiny is therefore uncertain.

The general desire to make the digest of our literature as complete as possible led to the adoption of a resolution by the American Ophthalmological Society in 1919, approving of our Year Book, suggesting its enlargement, and urging cooperation in its plan.

Then, at the meeting of the Section on Ophthalmology of the American Medical Association in 1921, a resolution was adopted appointing a committee, with instructions practically to determine the possibilities or advisability of publishing an International Year Book of Ophthalmology.

That committee, as finally enlarged, is composed of Drs. Black, Bordley, Jr., Derby, Fernandez, Feingold, Gifford, Gradle, Green, Jr., Howe, Jackson, Knapp, Lancaster, Parker, Pusey, de Schweinitz, Sweet, Tooke, Troncoso, Wiener, Wilder, Wilmer, Weeks, Wood and Ziegler. It will be noticed that nearly all are members of the Academy.

In order to ascertain whether such a plan would be at all acceptable, a circular letter was sent to members of the Ophthalmological Society of the United Kingdom. The response was prompt and gratifying. The question was taken up by the Council of British Ophthalmologists, which body informed us officially that it was "desirous to collaborate and to take its part in the editorial work of the publication."

Meanwhile, our Committee has learned through Professor Kiffner of Berlin, that the staff of the *Zentralblatt*, with some twenty odd other writers and teachers, will publish shortly what is practically a Year Book, as an enlarged and improved edition of Nagel's *Jahresbericht*, the various aspects of ophthalmology be-

ing there classified in some two hundred and sixty divisions and subdivisions. Such a work will naturally be considered a necessity by many in spite of the natural desire of English readers to have a Year Book in their own language. But the evident necessity everywhere now for a saving of energy, of time, and especially of cost, will undoubtedly help to solve embarrassing problems, and there is strong reason for hope, that in the rather near future an International Year Book of Ophthalmology will become a reality.

In view of the foregoing we respectfully recommend the following:

1. That this Committee be known in the future as the Committee on a National Bureau of Medical Research.

2. That the members already named of a committee of the Section of Ophthalmology of the American Medical Association, who are also members of the Academy of Ophthalmology and Oto-Laryngology, are hereby empowered to act also in the name of this Academy.

3. That this committee shall cooperate with similar committees of the American Medical Association and other organizations or individuals striving for the creation of a Department in our Government for the improvement of public health, the encouragement of research, and the advancement of higher standards of medical practice.

4. As the collections of bibliographies, in a form for convenient reference, is the first step in intelligent investigations, therefore this Academy hereby joins with the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association, in its approval of the establishment of an International Year Book of Ophthalmology, and hopes that after a time Year Books of Oto-Laryngology and other branches of medicine may also be established by the profession itself, thus laying the foundation for a National Bureau of Medical Research.

LUCIEN HOWE, CHAIRMAN
EMIL MAYER,
EDWARD B. HECKEL.

It was moved by Dr. LUTHER C. PETER of Philadelphia, that this report be received and referred to the Council, and that the Committee be continued. Motion seconded and carried.

REPORT OF THE COMMITTEE FOR THE INVESTIGATION OF IRITIS
AND IRIDOCYCLITIS

In the report rendered by this Committee at the 1921 meeting of the Academy at Philadelphia, it was suggested that, if the investigation of the etiology of iritis and iridocyclitis was to continue, there should be formed a new committee, the members of which should be located in large clinical centers, with the necessary laboratory facilities at hand to carry on an extensive and complete examination in each case, and that a member of the Committee should personally supervise the work. This suggestion was not adopted by the Academy, and the present committee was reappointed.

At a meeting of the Committee early in the year, it was agreed that the method of investigation hitherto employed was unsatisfactory, and that it was impossible to come to any definite and practical conclusion through the combined efforts of members of the Academy. The Committee further concluded that it was not practical for the Academy as a whole to carry on an investigation of this character.

We suggest that the Academy either (1) discontinue the investigation and discharge the Committee, or (2) establish a laboratory in one of the large clinical centers, to which patients may be sent and thoroughly investigated without inflicting the burden of what seems to the patient an unnecessary expense.

Respectfully submitted,

WILLIAM C. FINNOFF, CHAIRMAN

JOHN GREEN, JR.

WILLIAM L. BENEDICT.

It was moved by Dr. EDWARD B. HECKEL of Pittsburgh, that this report be received and referred to the Council. Motion seconded and carried.

REPORT OF COMMITTEE ON PUBLICITY AND SERVICE

The Committee on Publicity and Service created at the last annual session of our Academy, begs to submit its first report.

It may be wise to first enunciate the functions of this committee:

1. To publish authentic facts in the lay press relative to the activities of the profession.

2. To appeal to the Legislatures, with the approval of the Council, to secure the support of, or protest against, legislation.

3. To recommend, in addition to those measures already proposed, to aid the deaf, to restore defective speech and secure the teaching of proper enunciation, and to conserve and protect vision.

Quite a broad, farreaching, comprehensive and onerous undertaking, the importance of which needs no comments, as the necessity for such propaganda is selfevident.

The profession in the past has kept within itself and has failed to mingle with the public. So much so, that we have been frequently accused of being obstructionists, of sitting calmly by waiting for some adverse legislation, and then opposing it with might and main.

The profession should take the public into its confidence and begin a campaign of education. It should tell the public what certain cults are supposed to stand for, and what they really do stand for. It should tell the public what there is and what there is not in any new fancy, theory or fad. In short, the profession should be constructive. It should enter upon a campaign of publicity through the public press, which campaign must be carried on in a purely altruistic and unselfish manner, so as to impress the public with our real sincerity.

The public press is always interested in anything which might be called "news", but much of the matter which should be placed before the public for its own protection and welfare would not be regarded as "news" by the public press. The public press, too, has a style of its own, which is not easily imitated by the novice, so that our problem at once becomes difficult.

After some experience and considerable thought, we feel that the only way to succeed in this most important work is to employ a publicity man, one trained in newspaper work, through whom the public could be kept informed. Our thought is that the members of the Academy prepare the articles, and then have the publicity man rewrite them in newspaper style for publication, after the Committee on Publicity has approved them. This, too, is only part of the work. This same publicity man should keep the Committee informed on any proposed State or National legislation pertaining to the public health.

It goes without saying that such a plan, if we were to employ a man to devote all of his time to this work, would require the expenditure of considerable money. It is possible, however, to employ a man already in publicity work, on part time pay. We would suggest a man well fitted for this work, who may be already employed by some large corporation, a man who spends

much of his time at the National Capital, and who therefore could keep our Committee well informed at all times.

We therefore recommend to the Academy and to the Council the serious consideration of the employment of a publicity man, to act with and under the supervision of this Committee and the Council of this Academy.

Respectfully submitted,

EDWARD B. HECKEL, CHAIRMAN.

It was moved by Dr. GEORGE F. KEIPER that this report be received and referred to the Council. Motion seconded and carried.

REPORT OF SECTION ON INSTRUCTION

Last year marked the opening of a new phase of work in the Academy—the Section on Instruction. Under the direction of the Council, your Committee prepared a program of instruction covering three days. We had nearly 600 members registered for the course in Philadelphia, and they pronounced the three days' course a success. The Committee therefore was encouraged to continue it this year, but the Council limited the time this year to two days instead of three. We hope to carry on with as much success this year as last. Of course, this all depends upon the active cooperation of Council and the interest of the membership of the Academy at large, but if that is maintained at the same pitch as last year, we feel the Section on Instruction in the future will be a success.

There are several phases of the work I wish to speak of. A little closer cooperation between the Council and the Committee is essential. The Council is not and cannot be fully aware of all the actions of the Committee, nor can the Committee refer all its actions to the Council for approval, but there can be closer cooperation than there has been in the past.

This year, the same Committee is functioning as last, but in order to prevent apostolic succession, the Committee wishes to recommend that one member be retired automatically each year, and a member of the Academy be appointed by the outgoing President to fill his place. Thereby each member of the Committee will serve four years in turn. This of course must be referred to the Council, and is offered by the Committee merely as a suggestion.

From the standpoint of finance, last year the Committee came through with a balance of about \$100, and we found that there

were unnecessary expenses incurred in our ambition to present something rather unusual. We found we could limit the expense to a great extent, and we hope to reduce the cost of the Section on Instruction to members of the Academy to possibly half what it is now.

Furthermore, the Committee has purchased for the use of the Section on Instruction the latest model projection lantern, for the projection of opaque material as well as transparencies. This is the property of the Academy, held by the Committee on Instruction, and is for the use of the Academy during the meetings as well as by the Section during the year.

In conclusion, the Committee merely wishes to express its appreciation of the support which the members of the Academy have given this Section, and we trust we will be able to give you what you want in the future.

HARRY S. GRADLE, CHAIRMAN.

It was moved by Dr. LUTHER C. PETER that the report be received and referred to the Council for action. Motion seconded and carried.

REPORT OF SECTION ON OPHTHALMOLOGY AND OTO-LARYNGOLOGIC PATHOLOGY

To recapitulate what has been done—there has been an agreement entered into with the Army Medical Museum at Washington, whereby the Academy will endeavor to send in, from individual members, such specimens as may be valuable or may be of interest. These specimens will be sectioned and examined in Washington, the diagnosis corroborated by pathologists, and the diagnosis returned to the owner of the specimen; the specimen remains in the Army Medical Museum at Washington as a part of the permanent collection. Needless to say, this is entirely without expense to any of the parties concerned. So far, during the year we have received 95 eye specimens and two ear, nose and throat. Of the 95 eye specimens, we have 57 mounts now on exhibition in the room at the other end of this hall.

We have found the Section on Pathology a going concern. It is being extensively used by many men, and we feel that the time has now come to extend and nationalize the Section on Pathology in exactly the same manner as the Board of Ophthalmic Examiners is nationalized, and the Committee wishes to recommend to the Academy the following scheme: That the Academy divide the Section on Pathology into Eye; and Ear,

Nose and Throat. The two cannot go hand in glove. In this way we hope to interest a larger number of people throughout the country in sending in specimens, and to increase the interest in ophthalmic pathology. We believe there is a future for the ophthalmic pathologic museum now started in Washington, a future that will be of great value both to the beginner and to the established ophthalmologist who wishes to refresh himself by sending in pathologic specimens.

HARRY S. GRADLE, CHAIRMAN.

It was moved by Dr. EDWARD B. HECKEL that this report be received and referred to the Council. Motion seconded and carried.

REPORT OF AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS

The American Board for Ophthalmic Examinations held its twelfth examination in Millard Hall, Minnesota University. There have been certificated up to date 342 persons in this country. We certificated a number at the session last evening. Many of the prospective candidates for membership in the national societies are not well aware of just what this Board stands for. We observe that from the attitude some of the candidates take in approaching the Board.

Most of you are familiar with the fact that this movement was organized some seven or eight years ago, supported by the special societies, two of which can so control their membership that they can make certain requirements for eligibility in their Society. These are the American Ophthalmological Society, and this Academy of Ophthalmology and Oto-Laryngology, each of which is obliged in its By-laws and Constitution to require a certificate from this Board for eligibility to membership in the society. Membership in the Sections of the American Medical Association, either Ophthalmology or Oto-Laryngology, cannot be so controlled, because any member of the American Medical Association is equally entitled with all others to membership in any section. At the same time, the Section on Ophthalmology supports this movement.

Now this Academy of Ophthalmology and Oto-Laryngology is somewhat differently constituted from the American Ophthalmological Society, in that it has two branches of members—those who devote themselves exclusively to ophthalmology, those who devote themselves exclusively to oto-laryngology, and as we know, many who are compelled by circumstances to practice both

specialties. Now this Academy desires to control its rapidly growing membership, and it has subscribed to the idea of requiring applicants for membership to pass the examination of this Board and obtain its certificate of proficiency. Or if the individual does not care to do that, he may come through the door of Oto-Laryngology, and then he must pass examination by the committee which has been appointed on Oto-Laryngology. But unless these two boards cooperate well in their work, we can see that the very purpose of such a requirement falls down.

Then, again, it occurs to us that it might be well, and would be perfectly proper, for those who are practicing both specialties to be required to pass both boards, because if one board is not operating as actively as the other, you can see there would be a failure in the purpose of that requirement.

One thing we have observed, and that is that applicants for certificates of this American Board for Ophthalmic Examinations do not fully appreciate the importance of putting their applications in early, so that we have up to now at these different examinations used considerable leniency in the matter of allowing candidates to come before the Board with applications. Even yesterday, a number came without any preliminary application having been made. The Board feels now, however, that it has gone far enough in that matter, as its work is increasing very considerably each year. We have a larger number of examinations and our duties are considerably magnified, and the Board has therefore by vote made a ruling, that in the future, the published requirements of the Board will be strictly adhered to, and these published requirements, which can be obtained by application to the Secretary's office, provide that the individual must fill out an application blank and must comply with all the requirements of the Board before he will be admitted for examination. The reason for that is that this work is done by members scattered widely throughout the country, and these applications have to be sent from one to the other. When an individual applies for a certificate from the American Board, he fills out an application blank and sends it to the Secretary's office; this is sent to the members of the Requirements Committee, and they vote on what kind of examination that particular individual shall be subjected to. They determine whether his examination shall consist only of presentation of carefully prepared case records, or whether in addition to that he shall submit to a most comprehensive examination at some time and place where the Board may conduct an examination; and unless we allow time for that we cannot

accomplish our purpose. The Board has therefore determined by vote, that all candidates must present their credentials a long enough time in advance so that these requirements can be met, and in our circular of information, which the individual can obtain from the Secretary's office, we state that case reports must be sent to the Secretary at least sixty days before examination. If this is complied with, it makes the examinations more than satisfactory.

W. H. WILDER, FOR THE COMMITTEE.

It was moved by Dr. EDWARD B. HECKEL that the report be received and filed. Motion seconded by Dr. EMIL MAYER.

It was moved by Dr. GEORGE F. KEIPER of Lafayette, Indiana, as a substitute, that the report be received and adopted. Substitute accepted and motion carried.

REPORT OF COMMITTEE ON EXAMINATIONS IN OTO-LARYNGOLOGY

The Committee met, for the first time, last year in Philadelphia in a very perfunctory manner, and not all the committee were present. However, it performed its duties and examined a number of candidates for admission into this Academy.

Yesterday, the Committee met with practically every member present, and examined quite a large number of applicants. It was noticeable that quite a good deal of progress had been made in the work. During the year, we sent out to every member a questionnaire similar to that which the Ophthalmic Section sends out, and received some replies. Unfortunately, not enough time was allowed, so we could not send these replies to the different members of the Committee.

The Committee feels exactly as Doctor Wilder has expressed. The Oto-Laryngology Section does not want to stand one iota lower than the Ophthalmic Board. However, we have a different condition to contend with, in that we have five national societies instead of three—the American Otological, the American Laryngological, the Triological, the Section on Laryngology of the American Medical Association, and this Academy,—so it is a little more difficult. But the Committee has discussed arranging a mode of procedure, whereby we hope to have such a Board as the excellent American Board for Ophthalmic Examinations.

JOSEPH C. BECK, CHAIRMAN.

It was moved by Dr. EDWARD B. HECKEL that this report be received and referred to Council. Motion seconded and carried.

REPORT OF NECROLOGIST

The following deaths occurred during the year:

Dr. K. P. Battle.....	Raleigh, N. C.
Dr. C. D. Conkey.....	Duluth, Minn.
Dr. Alanson W. Hawley.....	Seattle, Wash.
Dr. L. H. Landman.....	Cincinnati, O.
Dr. H. L. Lynah.....	New York City
Dr. Fontaine B. Moore.....	Memphis, Tenn.
Dr. J. R. Robinson.....	Colorado Springs, Colo.
Dr. Charles C. Stuart.....	Cleveland, O.
Dr. C. W. Tangeman.....	Cincinnati, O.

The CHAIR requested that the Academy pay its respect to the memory of those who had died during the year by standing a moment in silence.

Business Session adjourned.

September 20, 1922.

The second Business Session was called to order at 9:10 a. m. by the President, who called for the Report of the Council.

REPORT OF THE COUNCIL

The Council places in nomination the following names:
 President, Thomas E. Carmody.....Denver, Col.
 1st Vice-President, Hunter H. McGuire....Winchester, Va.
 2nd Vice-President, John H. Morse.....Minneapolis
 3rd Vice-president, S. Hanford McKee.....Montreal, Can.
 Treasurer, Secord H. Large.....Cleveland, O.
 Secretary, Luther C. Peter.....Philadelphia
 Editor of Transactions, Clarence Loeb.....Chicago

COUNCIL

William R. Murray.....Minneapolis
 Joseph L. McCool.....Portland, Ore.

AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS.

Edward Jackson.....Denver (2 years)
 Lee M. Francis.....Buffalo (3 years)

OTO-LARYNGOLOGIC BOARD.

Joseph C. Beck.....	Chicago
Thomas E. Carmody.....	Denver
J. M. Ingersoll.....	Cleveland
Harris P. Mosher.....	Boston
Ross H. Skillern.....	Philadelphia

COMMITTEE ON POSTGRADUATE INSTRUCTION.

W. P. Wherry.....	Omaha, Neb. (4 years)
Harry S. Gradle.....	Chicago (3 years)
E. C. Ellett.....	Memphis (2 years)
Wm. R. Murray.....	Minneapolis (1 year)

The Council has passed the following resolutions relative to the Transactions:

1. Authors must place papers in the hands of the Secretary immediately after reading. It is necessary to enforce this ruling, because some manuscripts have been lost and a great deal of confusion has arisen because of nonreturn of manuscripts; consequently the Council feels that this ruling must be adhered to. Manuscripts of all papers read shall be placed in the hands of the Secretary immediately after delivery.

2. The Academy will pay one-half the cost of necessary illustrations, with the exception of colored plates, and all necessary illustrations must be published in the Transactions. Alterations in the proof, beyond a reasonable amount, must be paid for by the author. Unfortunately, the Editor of the Transactions says, many articles are entirely rewritten, which adds a great deal to the cost of publication of the Transactions, since it must be taken down and reset. So if a man wishes to alter his paper, he should be willing to pay for the alterations.

The Council has also made this ruling, that the Treasurer may, upon approval of Council, remit the dues of any member when there is a good reason brought forth why this should be done. The reason for this is that many times members of long standing in the Academy meet with disaster, or with illness, and according to our present rule they must be dropped. This makes it possible to retain in the membership of the Academy those who otherwise would have to be dropped for nonpayment of dues.

The next meeting place will be Washington, D. C.

Respectfully submitted,

LEE M. FRANCIS.

The President asked for further nominations from the floor. There being none, this report was held over until Thursday morning.

The following Amendment to the Constitution and By-Laws was then offered by the Secretary, Dr. LUTHER C. PETER.

Article 3, Section A, to be amended so as to read: "All applicants for membership must be approved by the American Board for Ophthalmic Examinations, or by the Oto-Laryngologic Board, designated by the Academy."

The reason for this change is to correct the equivocal phrase that exists at present. It was a question whether members who had not passed this Board were eligible for the program, and this change is proposed in order to eliminate that phase of it, making these requirements to apply only to incoming members. I move that this Amendment to the Constitution be adopted.

Motion seconded by Dr. JOSEPH C. BECK, and carried.

REPORT OF THE COMMITTEE ON THE STANDARDIZATION OF TUNING FORKS AND HEARING TESTS

At the meeting of the Academy held in Philadelphia, October, 1921, a motion was made by Dr. Horace Newhart of Minneapolis, to the effect that the Chair appoint a committee of three (this later increased to four) to cooperate with a committee from the American Otological Society, the American Triological Society, and the Section on Ear, Nose and Throat of the American Medical Association, whose duty it should be to standardize the instruments used for testing the acuity of hearing, the methods of their application, and the manner of the reporting the results of such tests. The Committee appointed consisted of Doctors L. W. Dean of Iowa City, Iowa; George W. Mackenzie of Philadelphia; E. G. Gill of Roanoke, Virginia, and Robert Sonnenschein of Chicago, Chairman.

So far as we have been able to learn, neither the Section on Ear, Nose and Throat of the American Medical Association nor the Triological Society have a committee on this subject, but we did confer with the committee of the American Otological Society, and through the courtesy of the Chairman, Dr. Edw. B. Dench of New York, we were furnished with a list of their recommendations. These were rather brief and were largely to the effect that a low standardized fork should be used, which will give a range of 26

up to 64 double vibrations. For testing bone conduction, either a small c^1 , 256, or a small c^2 , 512 double vibration fork, whichever is the better make; the ordinary Galton whistle for the determination of the upper tone limit, these instruments being recommended as the most available, even though not entirely accurate from the viewpoint of the physicist. The Committee proposed to have these instruments accurately made.

Written communications were had with the various members of our Committee, to obtain the views held regarding the forks used and the methods of their application in the various tests. A meeting was held in Washington, May 5, 1922, at which a number of points were tentatively decided upon, and the following recommendations made:

First: The Character of the Fork: The fork, if possible, should be well balanced, rustless (if such metal can be obtained), of a definite pitch, definite duration of vibrations, moderate in price, so made as to eliminate loud overtones and avoid rattling from the clamps holding the weights. In place of the projecting and easily broken thumbscrews to hold the weights on the prongs of the forks, one manufacturer uses round, flat, clamp like weights.

Second: Forks should be held uniformly near the ear, preferably with the wide surface of the prongs parallel with the ear, in order to get a maximum sound wave and avoid interference waves which occur at the nodal points.

Third: The Committee is not yet unanimous in opinion regarding the methods of stimulating the fork, or rather of exciting it, and also the method of timing the fork. It was not definitely determined whether it is better to note the duration of time during which the fork was heard, or to decide the degree of hearing by the distance from the ear at which the fork can be appreciated.

Fourth: The Committee recommends that at first only three standard tests be considered, namely: the Weber, the Schwabach, and the Rinne. With reference to the first two tests, no decision was reached regarding which fork should be used. The Committee thought one of the middle tones, approximately small c^1 , 256 double vibrations, or somewhat lower would do. For the Rinne test, a fork of middle tone, the exact pitch to be determined later, should be used. The time during which the fork is heard by bone and air conduction should be definitely designated. The construction of the fork ought to be such, that normally the difference between

bone and air conduction is about 30 to 40 seconds. When vibrating in the air alone, the fork should be heard approximately 100 seconds. This of course requires a rather expensive fork. Furthermore, the low limits can be determined possibly by a fork of 32 vibrations, adjustable to 54 double vibrations. The high limit should be determined either by means of the Galton whistle or the monochord.

Fifth: Regarding the forks recommended, there might first be a minimum list, namely: A low fork of 32 double vibrations, a medium fork of 256 double vibrations, and a high fork of 2,048 double vibrations per second. Secondly, a desirable list consists of about five forks: 32, 128, 256, 512 and 2,048 double vibrations per second.

Sixth: The Committee has been conferring with an expert manufacturer of standardized forks, and has also considered the feasibility or possibility of using rustless metal in the construction of the forks. This man in the East is experimenting with a rustless metal, and we are hoping within the few months to get some definite information regarding the possibility of having forks made of this particular metal. Where forks are nickeled to prevent rusting, there is a great disadvantage: first of all, the nickeling may interfere somewhat with the vibration of the fork; and secondly, when the nickel peels, adventitious sounds are produced by the vibration of the loose pieces of metal. On the other hand, if a fork is not nickeled and is not kept properly oiled, rusting invariably occurs. Therefore, we are greatly interested in the problem of obtaining a rustless metal.

Seventh: It is of course impossible for any committee in such a short space of time, or even in a longer time, to determine definitely what measures should be taken in so important a subject as the functional testing of the hearing. However, we are offering this brief and rather preliminary report in the hope that the subject will be thoroughly discussed on the floor of the meeting, and suggestions made to the Committee for their consideration. Finally, we would suggest that the Committee be continued for at least another year, in order to study the subject more in detail and submit further report at the next annual meeting.

Respectfully submitted,

GEORGE W. MACKENZIE,

E. G. GILL,

L. W. DEAN,

ROBERT SONNENSCHN, Chairman.

It was moved by Dr. EMIL MAYER that the report be accepted, placed on file, and the Committee continued. Motion seconded and carried.

REPORT OF COMMITTEE ON TENTH INTERNATIONAL CONGRESS OF OTOTOLOGY

As a delegate to the Tenth International Congress of Otolology, I beg to submit the following report:

The Congress was held in the Amphitheater of the Faculty of Medicine, University of Paris.

The Congress was opened according to schedule on Wednesday morning, July 19th, 1922, at nine o'clock, President Professor P. Sebileau in the chair. Your delegate, because of unavoidable train delay coming westward from Vienna, did not reach Paris on the opening day of the Congress, much to his regret. He reached the place of meeting on the morning of the second day, when he presented his credentials to the Assistant to the Secretary, who promised to forward them. He next registered and asked for a program of the meeting, when he was told that the programs for the day had not yet arrived from the printer. After some minutes of waiting, a batch of them were delivered. Each day's program was printed on a different colored pamphlet, copies of which are herewith submitted.

Lack of knowledge of the French language on the part of your delegate was a decided handicap. Incidentally, the suggestion is offered, that in the future, it would be well for the Academy in selecting a delegate to a Congress which is to be held in a foreign country, to choose one who is quite familiar with the particular language of that country.

The attendance at the Congress was small compared with that of ten years ago at Boston. Not more than sixty could be counted present at any session. The business proceedings were conducted in the French language. The five or six Americans present appeared to be no better off than your delegate. We all raised our hands when certain motions were put and carried. Following the business session at which this took place, your delegate conferred with the other Americans to learn from them what they had voted "Yes" on. When he learned from them that they did not know exactly what the motion meant, your delegate suggested that it was a motion to invite the Teutons to the next Congress, when

they agreed to this interpretation. Among the papers on the program to be presented by Americans were the following:

"Otitic Meningitis," by MacCuen Smith of Philadelphia.

"Treatment of Lung Abscess by Lavage, using Bronchoscopy," by H. H. Forbes of New York City.

"Blood Clot Dressing," by Geo. E. Davis of New York City.

"Skin Grafts in Radical Mastoid Operation," by Edward B. Dench of New York City.

"Operative Treatment of Brain Abscess," by Willis J. Eagleton of Newark, N. J.

Your delegate had the pleasure of hearing the papers of Drs. Dench, Eagleton and Davis read at the Congress, and wishes to congratulate them for their able presentation of their subjects.

On the whole, the Congress was a success, but not as successful from the standpoint of attendance and enthusiasm as that of the Ninth International Congress held at Boston, due no doubt to postwar conditions, and to the absence of the Germans and Austrians.

Professor Schmieglow was elected President for the next Congress, which is to be held at Antwerp ten years hence, when the German speaking people are expected to participate.

Respectfully submitted,

GEORGE W. MACKENZIE.

It was moved by DR. EMIL MAYER that the report be received with thanks, and the Committee discharged. Motion seconded and carried.

REPORT OF AUDITING COMMITTEE

Your Committee went over the books of the Treasurer and find them to be correct.

JOSEPH C. BECK,
WM. L. DAYTON.

It was moved by DR. C. D. WESTCOTT that this report be accepted and the Committee discharged. Motion seconded and carried.

Business Session adjourned.

SEPTEMBER 21, 1922.

The third Business Session was called to order at 9:10 a. m. by the President.

Under the head "Election of Officers," the Council presented its report of the previous day. It was moved by DR. EMIL MAYER that the Secretary cast the ballot of the Academy for the names mentioned in this report. Motion seconded and carried and ballot cast accordingly.

The Chair then announced the appointment of the following Committees by the Council:

NATIONAL MEDICAL RESEARCH LABORATORY:

Dr. Lucien Howe, Chairman
Dr. Emil Mayer
Dr. Edward B. Heckel

COMMITTEE ON PUBLICITY AND SERVICE:

Dr. E. C. Ellett, Chairman
Dr. Ross H. Skillern
Dr. Emil Mayer
Dr. H. W. Loeb
Dr. Secord H. Large

SECTION ON INSTRUCTION:

Dr. W. P. Wherry, 4 years.
Dr. Harry S. Gradle, 3 years.
Dr. E. C. Ellett, 2 years.
Dr. W. R. Murray, 1 year.

INVESTIGATION OF IRITIS AND IRIDOCYCLITIS:

Dr. John Weeks

AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS:

Dr. Lee M. Francis, 3 years.
Dr. Edward Jackson, 2 years.
Dr. Allen Greenwood, 1 year.

EXAMINATIONS IN OTO-LARYNGOLOGY:

Dr. Joseph C. Beck, Chairman
Dr. T. E. Carmody
Dr. John M. Ingersoll
Dr. Harris P. Mosher
Dr. Ross H. Skillern

SECTION ON OPHTHALMIC AND OTO-LARYNGOLOGIC PATHOLOGY:

Dr. Harry S. Gradle, Chairman
Dr. Ira Frank
Major Coupel, U. S. Army

The Secretary read the following names of applicants approved as Fellows in the Academy:

- Dr. Paul Morris Albright, Philadelphia.
- Dr. Neil Bentley, Detroit, Mich.
- Dr. Conrad Berens, Jr., New York City.
- Dr. Augustus Blickenstaff, Peoria, Ill.
- Dr. James C. Braswell, Tulsa, Okla.
- Dr. Henry H. Briggs, Asheville, N. C.
- Dr. Emesh W. Carpenter, Greenville, S. C.
- Dr. Joseph W. Charles, St. Louis.
- Dr. William C. Comee, Green Bay, Wis.
- Dr. Frederick A. Douglas, La Crosse, Wis.
- Dr. Harry D. Earl, Philadelphia.
- Dr. Nora M. Fairchild, Omaha, Neb.
- Lieut. Sherman B. Forbes, Annapolis, Md.
- Dr. John H. E. Fust, Erie, Pa.
- Dr. William W. Gailey, Bloomington, Ill.
- Dr. Raymond S. Goux, Detroit.
- Dr. John F. Hardesty, St. Louis.
- Dr. Arthur W. Herbert, Philadelphia.
- Dr. Wilber L. Hopper, Fort Scott, Kan.
- Dr. V. Reeves Hurst, Longview, Texas.
- Dr. Arnold B. Kauffman, Chicago.
- Dr. Joseph J. Kemler, Baltimore, Md.
- Dr. Stanley E. Kerrick, Minneapolis.
- Dr. Morton J. Keys, Victoria, B. C.
- Dr. Barney M. Kully, Omaha, Neb.
- Dr. Claude L. LaRue, Boulder, Col.
- Dr. John E. Loftus, Philadelphia.
- Dr. Clara A. March, Buffalo, N. Y.
- Dr. George J. McKee, Pittsburgh.
- Dr. William F. Moore, Philadelphia.
- Dr. Harry E. Oesterling, Wheeling, W. Va.
- Dr. Clarence N. Peeler, Charlotte, N. C.
- Dr. Clarence P. Rice, Wahpeton, N. D.
- Dr. James A. M. Russell, Erie, Pa.
- Dr. Charles E. Savery, South Bend, Ind.
- Dr. Francis X. Siegel, Cincinnati, O.
- Dr. Fred S. Stahlman, Charleroi, Pa.
- Dr. Albert L. Steinfield, Toledo, O.
- Dr. Frank M. Sulzman, Troy, N. Y.
- Dr. Grover C. Todd, Pittsburgh, Pa.
- Dr. Robert S. G. Welch, Philadelphia.
- Dr. Charles P. White, Wilmington, Del.
- Dr. George E. Winter, Jackson, Mich.

It was moved by Dr. EMIL MAYER that those who have been recommended by both Boards and by the Council as Fellows shall be elected and declared members of the Academy. Motion seconded and carried.

REPORT OF COMMITTEE ON PRESIDENT'S ADDRESS.

Your committee has carefully considered the Address of the President and fully coincides with the views and recommendations therein expressed.

We recommend the appointment of a committee of five—one of whom shall be our President—to present a report at the next meeting of the Academy on the proper relation of the teaching of ophthalmology, otology and laryngology to the general undergraduate curriculum, and also to present the views here endorsed before other organizations considering this subject.

EDWARD JACKSON

GEORGE F. KEIPER

CASSIUS D. WESCOTT, CHAIRMAN.

Moved by Dr. WESCOTT that this report be approved. Motion seconded by Dr. EMIL MAYER and carried.

The President announced that committee mentioned above would consist of the members making the report, the President, and one additional member.

COMMITTEE ON PRESIDENT'S ADDRESS.

Dr. Walter R. Parker

Dr. Cassius D. Westcott

Dr. Edward Jackson

Dr. George F. Keiper

Dr. Harris P. Mosher.

INSTALLATION OF OFFICERS:

Dr. PARKER requested Dr. Ellett to escort Dr. Carmody, the newly elected President, to the stage.

Dr. THOMAS E. CARMODY: Mr. President, Members of the Academy: I cannot tell you how much I appreciate this honor. I wish to thank the Council and the members of the Academy for this great honor. I consider that there are many other men in the Academy who could have filled the place much better than I will be able to, but I will do the best I know how and give you all the service possible. (Prolonged applause.)

DR. PARKER: The work of the local committee is well known to all of you. Dr. Newhart has worked his head off and, more than that, has made the rest of the committee work also, and the result as you know has been wonderful. I shall be glad to entertain a vote of thanks to this committee.

DR. EMIL MAYER: I move that this Academy extend its most cordial and sincere thanks to our fellow members and the members of the profession in the Twin Cities for their wonderful hospitality, which we most cordially appreciate.

DR. JACKSON: I wish to second the motion, but also wish to call attention to an error in the Chairman's remarks. Dr. Newhart has worked very hard but he has not worked his head off; the arrangements have been perfect in every detail. (Laughter and applause.)

The motion was put and carried by a rising vote.

DR. PARKER: Before we proceed to the program I wish to take just a moment to express to you my thanks for the great consideration you have shown me. We have not quite lived up to the program but we have done our best, and I appreciate your courtesy and help. (Applause.)

No further business presenting, the Business Session adjourned.

Members are requested to notify the secretary of any error or omission in names, addresses or specialties; also of any change in address before the time of the 1923 meeting.

ALPHABETIC LIST

Honorary Fellows

- ELLIOT, LT.-COL. R. H.,
54 Welbeck St., Cavendish Sq., London, W., Eng.
DUNDAS-GRANT, SIR JAMES,
148 Harley St., W. 1, London, Eng.
GRAY, ALBERT A.....Glasgow, Scotland
JOHNSON, LINDSAY..Britannia Bldg., West St., Durban, Natal
ELSCHNIG, PROF. A.
Ferdinandstrasse, 10, Prague, Czecho-Slovakia
FERNANDEZ, JUAN SANTOS.....Prado, 105, Havana, Cuba
LAWFORD, J. B.,
99 Harley St., Cavendish Sq., W., London, Eng.
STEPHENSON, SYDNEY,
24 Thayer St., Manchester Sq., London, Eng.
VAN DER HOEVE, PROF. J.,
Rijnsburgerweg, 6 A, Leiden, Holland

Life Members

- *DAYTON, W. L.....Funke Bldg., Lincoln, Neb., OALR
*FOSTER, HAL.....Altman Bldg., Kansas City, Mo., ALR
HOWE, LUCIEN....520 Delaware Ave., Buffalo, N. Y., Op.

Fellows

- *ABBOTT, W. J.....628 Union Bldg., Cleveland, O., OALR
ADAMS, A. L....323 West State St., Jacksonville, Ill., OALR
*ADAMS, C.....52 West State St., Trenton, N. J., OALR
ADAMS, DANIEL S.,
520 Hume-Mansur Bldg., Indianapolis, Ind., ALR
ADAMS, ELDRIDGE S..Moore Bldg., San Antonio, Tex., OALR
*ADAMS, FRED'K W...Roosevelt Clinic, Seattle, Wash., OALR
*AGNEW, FRED'K W.,
120 3rd Ave., S.*E., Independence, Ia., OALR
AINSLEE, GEORGE,
606 Oregonian Bldg., Portland, Ore., OALR
ALBRIGHT, P. M.,
League Island Naval Hosp., Philadelphia, Pa., ALR
ALDEN, ARTHUR M., Walter Reed U. S. A. Gen. Hosp.,
Washington, D. C., ALR
ALDERDYCE, W. W.,.....Nasby Blk., Toledo, O., OALR
ALKIRE, H. L.....614 Kansas Ave., Topeka, Kan., OALR
ALLEN, CHAS. E.,
712 Waldheim Bldg., Kansas City, Mo., OALR
ALLEN, G. H.....Mills Bldg., Topeka, Kan., OALR
ALLEN, GEORGE V.
1434 Harrison Bldg., Topeka, Kan., OALR

*Attended 1922 Meeting.

- ALLEN, J. H.....Majestic Bldg., Denver, Colo., ALR
 ALLEN, L. P.....19 Jefferson Ave., Oshkosh, Wis., OALR
 ALLEN, S. E.....22 West 7th St., Cincinnati, O., ALR
 ALLPORT, F.....7 West Madison St., Chicago, Ill., OALR
 ALTER, F. W.....Colton Bldg., Toledo, O., OALR
 ALTRINGER, ARTHUR N.,
 1100 Rialto Bldg., Kansas City, Mo., ALR
 *ALWAY, ROBERT D.,
 423 S. Lincoln St., Aberdeen, S. D., OALR
 AMBERG, EMIL,
 David Whitney Bldg., Detroit, Mich., ALR
 ANDERSON, W. B....507 Main Ave., Brownwood, Tex., OALR
 *ANDREWS, A. H.....Reliance Bldg., Chicago, Ill., OALR
 *ANDREWS, B. F.:25 E. Washington St., Chicago, Ill., OALR
 ANDREWS, H. D.....23 Allen St., Buffalo, N. Y., Op.
 ANDREWS, M. P...Dempsey Bldg., Manitowoc, Wis., OALR
 APPLEMAN, L. F.,
 308 South 16th St., Philadelphia, Pa., Op.
 *ARBUCKLE, M. F.....Lister Bldg., St. Louis, Mo., ALR
 ARNOLD, F. J.....182 Pearl St., Burlington, Vt., OALR
 *ARRELL, WILLIAM...317 Main E., Hamilton, Ontario, OALR
 ARROWSMITH, H.....170 Clinton St., Brooklyn, N. Y., ALR
 ASH, GARRETT G.....1 Main St., Bradford, Pa., OALR
 ASHLEY, T. W.....466 Park Ave., Kenosha, Wis., OALR
 ATKINSON, D. T.,
 1515 Russell Bldg., San Antonio, Tex., OALR
 AUERBACH, J.,
 120 W. 86th St., New York City, N. Y., ALR
 AUFMWASSER, H.....Cincinnati, O., OALR
 AUTEN, F. E.,
 1st National Bank Bldg., Belleville, Ill., OALR
 AVERY, F. T.....357 W. 63rd St., Chicago, Ill., Op.LR
 AYRES, W. McL.....4 W. 7th St., Cincinnati, O., Op.
 AYNSWORTH, H. T.....Amicable Bldg., Waco, Tex.; OALR
 *BAHN, CHAS. A.,
 722 Maison Blanche Annex, New Orleans, La., Op.
 BAILEY, F. W.....Security Bldg., Cedar Rapids, Ia., OALR
 *BAILEY, H.....840 Landers Bldg., Springfield, Mo. OALR
 BAIRD, CHAS. G.....Beatrice, Neb., OALR
 BAKER, C. H.....Crapo Blk., Bay City, Mich., OALR
 BAKER, H. B.....59 Main St., Taunton, Mass., OALR
 BALDWIN, KATE W.,
 1117 Spruce St., Philadelphia, Pa., ALR
 BALENTINE, P. L.,
 1524 Chestnut St., Philadelphia, Pa., Op.
 BALL, J. MOORES.....4500 Olive St., St. Louis, Mo., Op.
 BALL, M. V.....214 Penn Ave., W., Warren, Pa., OALR
 BALLENGER, H. C.,
 25 E. Washington St., Chicago, Ill., ALR
 BALLIN, M. J...108 E. 60th St., New York City, N. Y., ALR

- BANE, WM....330 Metropolitan Bldg., Denver, Colo., OALR
 *BANE, W. C.....Metropolitan Bldg., Denver, Colo., OALR
 *BANISTER, J. M.,
 400 Brandeis Theater Bldg., Omaha, Neb., OALR
 BARBER, F.....259 Alexander St., Rochester, N. Y., Op.
 BARCK, C.....Humboldt Bldg., St. Louis, Mo., Op.A
 BARDENHEIER, F. G. A...Frisco Bldg., St. Louis, Mo., ALR
 BARKAN, A.....77 Battery St., San Francisco, Cal., OALR
 BARKAN, H....Physicians Bldg., San Francisco, Cal., Op.
 *BARKER, CHAS. B.
 224½ W. Oklahoma Ave., Guthrie, Okla., OALR
 BARKER, O. G. A.,
 Johnstown Trust Co., Johnstown, Pa., Op.
 BARNES, L. E...6860 South Halsted St., Chicago, Ill., OALR
 *BARNHILL, J. F....Pennway Bldg., Indianapolis, Ind., ALR
 *BARLOW, ROY A.,
 416 Farmers Trust Bldg., Portland, Me., ALR
 BARON, F. S.....Peoples Bank Bldg., Zanesville, O., OALR
 BARTHOLOMEW, A. C.
 1020 Harrison St., Ft. Wayne, Ind., OALR
 BAUM, H. L.....Metropolitan Bldg., Denver, Colo., ALR
 BEACH, S. J.....704 Congress St., Portland, Me., OALR
 BEANE, GEORGE W.,
 Jenkins Arcade Bldg., Pittsburgh, Pa., OALR
 *BEATTIE, W. HENRY....252 Genesee St., Utica, N. Y., OALR
 *BEATTIE, ROBERT,
 David Whitney Bldg., Detroit, Mich., OALR
 BEATTY, H. G.....150 E. Broad St., Columbus, O., OALR
 BEAUDOUX, H. A.,
 401 La Salle Bldg., Minneapolis, Minn., Op.A.
 BECK, F. L.....408 Hynds Bldg., Cheyenne, Wyo., OALR
 *BECK, J. C.....2551 N. Clark St., Chicago, Ill., ALR
 BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
 *BEDELL, A. J.....344 State St., Albany, N. Y., Op.A
 BEEBE, C. S.....Wells Bldg., Milwaukee, Wis., OALR
 *BEGGS, W. F.....2 Lombardy St., Albany, N. Y., OALR
 BEIL, J. WALLACE,
 316 Argyle Bldg., Kansas City, Mo., OALR
 BELL, GEO. HUSTON,
 40 E. 41st St., New York City, N. Y., Op.
 BELL, M. H.
 First Natl. Bank Bldg., Vicksburg, Miss., OALR
 *BELLIN, JOSEPH,
 322 N. Washington St., Green Bay, Wis., OALR
 BELLOWES, GEORGE E...Rialto Bldg., Kansas City, Mo., Op.
 BEEM, I. F.....31 N. State St., Chicago, Ill., OALR
 *BENEDICT, W. L.....Mayo Clinic, Rochester, Minn., Op.
 BENETTO, FREDERICK R.,
 172 Woolwich St., Guelph, Ont., Canada, OALR
 *BENNETT, A. G.....26 Allen St., Buffalo, N. Y., Op.

- *BENSON, GEORGE E.,
1111 Metropolitan Bank Bldg., Minneapolis, Minn., OALR
- *BENTLEY, NEIL,
1161 David Whitney Bldg., Detroit, Mich., OALR
- BERENS, CONRAD, JR.,
9 E. 46th St., New York City, N. Y., Op.
- *BERGERON, J. Z...104 S. Michigan Blvd., Chicago, Ill., ALR
- BERNATZ, C. F.....Park Bldg., Pittsburgh, Pa., Op.
- *BERNSTEIN, E. J.....Kresge Bldg., Detroit, Mich., OALR
- BERRY, D. F..Hume-Mansur Bldg., Indianapolis, Ind., ALR
- BETTISON, D. L.....502 Wilson Bldg., Dallas, Tex., OALR
- BEYER, LOUIS J...449 Delaware Ave., Buffalo, N. Y., ALR
- BIGELOW, I. N...124 Waterman St., Providence, R. I., ALR
- *BINGER, H. E....1027 Lowry Bldg., St. Paul, Minn., OALR
- *BINKLEY, R. S.,
550 Fidelity Medical Bldg., Dayton, O., OALR
- *BISHOP, C. W.,
616 LaSalle Bldg., Minneapolis, Minn., OALR
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- *BLACK, N. M.....Wells Bldg., Milwaukee, Wis., Op.
- *BLACK, W. D.....Metropolitan Bldg., St. Louis, Mo., OALR
- BLACKBURN, W. J....661 Reibold Bldg., Dayton, O., OALR
- BLAIR, W. W..Diamond Bank Bldg., Pittsburgh, Pa., Op.
- BLAKE, W. F....516 Sutter St., San Francisco, Cal., Op.
- BLAKESLEY, T. S....Lathrop Bldg., Kansas City, Mo., OALR
- *BLEDSE, R. W...1005 Madison Ave., Covington, Ky., OALR
- *BLICKENSTAFF, A. J.,
804 Peoria Life Bldg., Peoria, Ill., OALR
- BLISS, C. B.....411 Columbus Ave., Sandusky, O., OALR
- BLUE, J. B.....Exchange Bldg., Memphis, Tenn., OALR
- BLUM, H. N.,
Maison Blanche Bldg., New Orleans, La., Op.
- BOBB, E. V.....1st Natl. Bk. Bldg., Mitchell, S. D., OALR
- BOECKMANN, EGIL...Lowry Bldg., St. Paul, Minn., OALR
- BOEHRINGER, H. W.,
1811 S. 22nd St., Philadelphia, Pa., Op.
- BOERNER, MORRIS H.,
Scarborough Bldg., Austin, Tex., OALR
- *BOILER, W. F.....426 S. Dodge St., Iowa City, Ia., OALR
- BONE, H. D.....29 Parker St., Gardner, Mass., OALR
- BONNER, H..840 Fidelity Medical Bldg., Dayton, O., OALR
- *BOONE, J. C.....315 Dean Bldg., South Bend, Ind., OALR
- *BOOT, G. W.....800 Davis St., Evanston, Ill., OALR
- BORDLEY, JAMES, JR.,
330 N. Charles St., Baltimore, Md., Op.
- BOUCHER, R. B....414 Banks Bldg., Vancouver, B. C., OALR
- BOURBON, OLIVER P.,
711 Lathrop Bldg., Kansas City, Mo., OALR
- *BOUVY, HARRY..New Foley Bldg., La Grande, Ore., OALR
- *BOYCE, G. H....408 E. D. St., Iron Mountain, Mich., OALR

- *BOYCE, WM. B..1019 Ludington St., Escanaba, Mich., OALR
 BOYD, E. T.....823 Majestic Bldg., Denver, Colo., OALR
 BOYD, G.....167 Bloor St., Toronto, Can., ALR
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 716 L. M. S. Bldg., South Bend, Ind., ALR
 BRADFIELD, J. A. L.,
 State Bank Bldg., La Crosse, Wis., OALR
 *BRASWELL, C. J.....726 Mayo Bldg., Tulsa, Okla., OALR
 BRAUNLIN, WM. H.
 Marion Natl. Bk. Bldg., Marion, Ind., OALR
 BRAWLEY, F. E...Michigan Blvd. Bldg., Chicago, Ill., OALR
 BRAWLEY, R. V....Wallace Bldg., Salisbury, N. C., OALR
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 BREWER, JOHN D...618 Main St., Dyersburg, Tenn., OALR
 BRIBACH, E. J....212 Simpson Bldg., Atchison, Kan., OALR
 *BRICKLEY, D. W.....196 W. Center St., Marion, O., OALR
 *BRIGGS, FRANCIS W.,
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 BROOKS, E. B....1010 Terminal Bldg., Lincoln, Neb., OALR
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 BROSE, L. D.....501 Upper First St., Evansville, Ind., OALR
 BROWN, C. M.....510 Delaware Ave., Buffalo, N. Y., ALR
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 BROWN, E. T.....30 Church St., Burlington, Vt., OALR
 BROWN, H. A..719 St. Paul Bldg., San Francisco, Cal., Op.
 BROWN, J. E.....239 East Town St., Columbus, O., OALR
 BROWN, JOHN C...632 Lowry Bldg., St. Paul, Minn., OALR
 BROWN, J. M....Brockman Bldg., Los Angeles, Cal., ALR
 BROWN, L. E.....2nd Natl. Bank Bldg., Akron, O., OALR
 BROWN, MORROW D.,
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 BROWN, M. G...802 University Bldg., Syracuse, N. Y., ALR
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 *BROWN, W. H.,
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 *BRUÉRE, G. E.....Journal Bldg., Portland, Ore., OALR
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 BULSON, A. E., JR.,
 406 W. Berry St., Ft. Wayne, Ind., OALR
 *BURCH, F. E.....Hamm Bldg., St. Paul, Minn., A
 BURKE, T. A.....446 Rose Bldg., Cleveland, O., OALR
 BURLESON, J. H.,
 Central Trust Bldg., San Antonio, Tex., OALR
 BURNS, S. S.....Humboldt Bldg., St. Louis, Mo., ALR
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 CALDWELL, R.,
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 CALHOUN, F. P.....Candler Bldg., Atlanta, Ga., OALR
 *CALLFAS, W. F....567 Brandeis Bldg., Omaha, Neb., ALR
 CAMERON, WILLIAM G.,
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 *CAMP, W. E.
 910 Donaldson Bldg., Minneapolis, Minn., OALR
 *CAMPBELL, C. H.,
 Columbus State Bk. Bldg., Columbus, Neb., OALR
 CAMPBELL, D. A.....David Whitney, Detroit, Mich., OALR
 *CAMPBELL, D. M....Peter Smith Bldg., Detroit, Mich., OALR
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 CAPRON, F. P.....118 Angell St., Providence, R. I., OALR
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 201 E. North St., Greenville, S. C., ALR
- CARR, G. W....30 S. Franklin St., Wilkes-Barre, Pa., OALR
- CARROLL, F.....Ely Blk., Cedar Rapids, Ia., OALR
- CARROLL, G. G...614 Main St., West, Rochester, N. Y. OALR
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- CARTER, W. W.,
 2 West 67th St., New York City, N. Y., ALR
- CARVILL, LIZZIE M..101 Newbury St., Boston, Mass., Op.
- CARY, E. H....Southwestern Life Bldg., Dallas, Tex., OALR
- CARY, G. C.....Grand Junction, Colo., OALR
- *CAVANAUGH, J. A...7 W. Madison St., Chicago, Ill., ALR
- *CAYCE, E. B.....Hitchcock Bldg., Nashville, Tenn., OALR
- CHADWICK, IRA B.,
 4 Terminal Bldg., Coffeyville, Kan., OALR
- CHAMBERS, T. R..15 Exchange Pl., Jersey City, N. J., OALR
- *CHAPMAN, V. A.....Wells Bldg., Milwaukee, Wis., OALR
- CHARLES, J. W.....Humboldt Bldg., St. Louis, Mo., Op.
- CHARLTON, C. C.,
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- *CHASE, E. FRANK...1008 Cobb Bldg., Seattle, Wash., OALR
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 377 Commonwealth Ave., Boston, Mass., ALR
- CHENEY, F. E.,
 64 Commonwealth Ave., Boston, Mass., Op.
- CHENEY, JAMES W.,
 702 Schweiter Bldg., Wichita, Kan., OALR
- *CHILDS, H. A.....Eagle Blk., Creston, Ia., OALR
- *CLARK, F. T.....92 Broad St., Westfield, Mass., OALR
- *CLARK, H. S....Syndicate Bldg., Minneapolis, Minn., OALR
- CLARK, I. G.....188 E. State St., Columbus, O., OALR
- *CLARK, J. S.....67 Stephenson St., Freeport, Ill., OALR
- CLARK, L. H.....337 Monroe Ave., Rochester, N. Y., OALR
- CLAY, G. E.....426 Candler Bldg., Atlanta, Ga., OALR
- CLEMENT, C. C.....Venetian Bldg., Chicago, Ill., OALR
- CLEVENGER, W. F.,
 Newton Claypool Bldg., Indianapolis, Ind., ALR
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- COCHRAN, R. W...West and 3rd Sts., Madison, Ind., OALR
- *COFFELT, THEODORE A.,
 Woodruff Bldg., Springfield, Mo., OALR

- COGAN, J. E.....Rose Bldg., Cleveland, O., OALR
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 COHEN, SAMUEL....1626 Spruce St., Philadelphia, Pa., ALR
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 COLLINS, H.....New Jersey Bldg., Duluth, Minn., OALR
 COLLINS, J. D.....90 Main St., Northampton, Mass., ALR
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 182 Manchester St., Battle Creek, Mich., OALR
 *COMEE, W. C.....Bellin Bldg., Green Bay, Wis., OALR
 *CONLON, F. A....Bay State Bldg., Lawrence, Mass., OALR
 *CONNOR, CHAS. E.....Lowry Bldg., St. Paul, Minn., OALR
 *CONNOR, RAY.....1410 Stroh Bldg., Detroit, Mich., OALR
 COOK, S. E.....Richards Blk., Lincoln, Neb., OALR
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 CRAFT, KENNETH L.,
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 CRANE, J. WELLINGTON.128 Third St., Trenton, N. J., ALR
 CRAWFORD, J. H.....Grant Bldg., Atlanta, Ga., OALR
 CREIGHTON, W. J.,
 1905 Chestnut St., Philadelphia, Pa., Op.
 *CRISP, W. H.....Metropolitan Bldg., Denver, Colo., Op.
 CROCKETT, R. L.....Oneida, N. Y., Op.
 CROSKEY, JOHN WELSH,
 1909 Chestnut St., Philadelphia, Pa., OALR
 CROSS, A. E.....Slater Bldg., Worcester, Mass., Op.
 CROSS, G. H.....525 Welsh St., Chester, Pa., Op.
 CROSSLEY, E. R.....29 E. Madison St., Chicago, Ill., OALR
 CRUMP, J. F.,
 507 Citizens Bank Bldg., Pine Bluff, Ark., OALR
 CULBERTSON, L. R....Masonic Temple, Zanesville, O., OALR
 CULLOM, MARVIN, McT.,
 208 Hitchcock Bldg., Nashville, Tenn., OALR
 CURDY, R. J....1134 Rialto Bldg., Kansas City, Mo., Op.
 CURRAN, EDW. J.,
 401 Waldheim Bldg., Kansas City, Mo., Op.

- CURRY, G. E....Westinghouse Bldg., Pittsburgh, Pa., Op.
 *CURTIS, W. L.,
 612 Security Mutual Bk. Bldg., Lincoln, Neb., OALR
 CUTLER, FRANKLIN E.,
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 *DAVIS, HARRY B.611 Lathrop Bldg., Kansas City, Mo., OALR
 DAVIS, J. E.....Kress Bldg., McAlister, Okla., OALR
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 DAYTON, G. O.....Lewisohn Bldg., Butte, Mont., OALR
 *DEAN F. W. National Bank Bldg., Council Bluffs, Ia., OALR
 DEAN, L. W.....12 S. Clinton St., Iowa City, Ia., OALR
 DEAN, WALTER.....Starks Bldg., Louisville, Ky.
 DEBOE, M. P.....621 Eaton St., Key West, Fla., OALR
 DECHERD, H. B.....Wilson Bldg., Dallas, Tex., OALR
 DECKER, J. C.Francis Bldg., Sioux City, Ia., OALR
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 2028 Chestnut St., Philadelphia, Pa., OALR
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 DENNIS, F. L...Burns Bldg., Colorado Springs, Colo., ALR
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 LYNCH, R. C.....Maison Blanche Bldg., ALR
 SMITH, V. C.....Maison Blanche Bldg., Op.
 TAQUINO, G. J.....Maison Blanche Bldg., ALR
 WEIL, A. L.....717 Maison Blanche Annex Bldg., ALR

SHREVEPORT

MOISE, A. B.....Commercial Bk. Bldg., ALR
 SCALES, J. L.....Commercial Bk. Bldg., OALR

MAINE

AUGUSTA

TURNER, O. W.....Augusta Trust Bldg., OALR

HOULTON

MITCHELL, FREDERICK W.....Fogg Bldg., OALR

PORTLAND

BARLOW, R. A.....Farmers Trust Bldg., ALR
 BEACH, S. J.....704 Congress St., OALR
 GILBERT, F. Y.....148 Park St., Op.A
 HASKELL, A. D.....145 High St., Op.
 HOLT, E. E., Jr.....723 Congress St., Op.A
 SPALDING, J. A.....627 Congress St., Op.A

MARYLAND

ANNAPOLIS

FORBES, LT. S. B.....U. S. N. Hosp., OALR

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., Op.
 FRIEDENWALD, HARRY.....1029 Madison Ave., Op.A
 GOLDBACH, L. J.....322 N. Charles St., OALR
 KEMLER, JOSEPH J.....1908 Eutaw Pl., OALR
 PETERMAN, H. F.....518 N. Charles St., OALR
 TARUN, WILLIAM605 Park Ave., Op.A
 WOODS, HIRAM842 Park Ave., Op.A

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
 ROBINSON, H. T.....Medical Bldg., OALR
 SHARRETT, GEORGE O.....119 Bedford St., OALR

MASSACHUSETTS

BOSTON

BRYANT, A. G.....	502 Beacon St.
CARVILL, LIZZIE M.....	101 Newbury St., Op.
CHENERY, W. E.....	377 Commonwealth Ave., ALR
CHENEY, F. E.....	64 Commonwealth Ave., Op.
FREEDMAN, L. M.....	419 Boylston St., ALR
GREENWOOD, ALLEN	82 Commonwealth Ave., Op.
HURLEY, E. D.....	419 Boylston St., Op.
HURLEY, E. H.....	419 Boylston St., Op.
INGLIS, H. J.....	Bay State Bldg., ALR
LANCASTER, W. B.....	520 Commonwealth Ave., Op.
LEMOINE, A. L.....	233 Charles St., Op.
LEWIS, J. P.....	543 Boylston St., ALR
MOSHER, H. P.....	828 Beacon St., ALR
RICE, G. B.....	293 Commonwealth Ave., ALR
SHANAHAN, T. J.....	419 Boylston St., ALR
STANDISH, MYLES.....	51 Hereford St., Back Bay, Op.
STEVENS, H. B.....	522 Commonwealth Ave., Op.
SUFFA, G. A.....	Gulford Hall, Op.
THOMPSON, P. H.....	308 Commonwealth Ave., Op.
TINGLEY, LOUISA P.....	9 Massachusetts Ave., Op.
TOBEY, G. L.....	416 Marlborough St., ALR
WELLS, D. W.....	The Westminster, Op.
WHITE, L. E.....	390 Commonwealth Ave., ALR

CLINTON

MONAHAN, J. A.....	181 Chestnut St., OALR
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FALL RIVER

RYDER, D. R.....	151 Rock St., ALR
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FRAMINGHAM

JESSAMAN, L. W.....	30 Hollis St., OALR
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GARDNER

BONE, H. D.....	29 Parker St., OALR
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HOLYOKE

HUSSEY, E. J.....	276 High St., Op.LR
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LAWRENCE

CONLON, F. A.....	Bay State Bldg., OALR
MERRILL, W. H.....	Bay State Bldg., OALR

LYNN

COBB, C. M.....	10 Nahant St., OALR
JONES, E. W.....	44 Atlanta St., Op.A
O'REILLY, W. F.....	44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....	278 Union St., OALR
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NORTH ADAMS

THOMPSON, G. H.....	18 Ashland St., OALR
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NORTHAMPTON

COLLINS, J. D.....	90 Main St., ALR
DOW, F. E.....	4 West St., Op.

PALMER

MOORE, G. A.....	Bank Bldg., OALF
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SALEM

HENNESSEY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....4 Chestnut St., OALR

CARLETON, RALPH.....10 Temple St., Op.

FRELIGH, C. A.....75 State St., ALR

IRWIN, V. J.....389 Main St., OALR

IRWIN, V. J., JR.....389 Main St., OALR

TAUNTON

BAKER, H. B.....59 Main St., OALR

WESTFIELD

CLARK, F. T.....52 Broad St., OALR

WORCESTER

CAHILL, J. W.....390 Main St., OALR

CROSS, A. E.....Slater Bldg., Op.

ESTABROOK, CHARLES.....390 Main St., OALR

MICHIGAN

ANN ARBOR

SLOCUM, GEORGE.....328 E. Huron St., Op.

BATTLE CREEK

COLVER, B. N.....182 Manchester St., OALR

FARNESWORTH, M. A.....146 Greenwood St., OALR

HAUGHEY, WILFRID.....24 W. Main St., OALR

SLEIGHT, R. D.....Post Bldg., OALR

STEGMAN, L. B.....Sanitarium, Op.

WENCKE, C. G.....16 Wentworth Ct., OALR

BAY CITY

BAKER, C. H.....Crapo Block, OALR

DETROIT

AMBERG, EMILDavid Whitney Bldg., ALR

BEATTIE, ROBERTDavid Whitney Bldg., OALR

BENTLEY, NEIL1161 David Whitney Bldg., OALR

BERNSTEIN, E. J.....Kresge Bldg., OALR

CAMPBELL, D. A.....David Whitney Bldg., OALR

CAMPBELL, DON M.....Peter Smith Bldg., OALR

CONNOR, RAY.....1410 Stroh Bldg., OALR

DEFNET, WM. A.....62 Adams St., W., ALR

GOUX, R. S.....545 David Whitney Bldg., OALR

HARTZ, H. J.....27 E. Adams St., ALR

MAIRE, L. E.....Park Bldg., Op.A

MERCER, R. E.....David Whitney Bldg., LR

MINER, S. C.....1564 Woodward Ave., ALR

ODELL, ANNA.....32 Adams St., Op.AL

PARKER, WALTER R.....David Whitney Bldg., Op.

RENAUD, G. L.....Fine Arts Bldg., OALR

SANDERSON, H. H.....David Whitney Bldg., OALR

SHURLEY, B. R.....62 W. Adams St., ALR

SIMPSON, H. L.....David Whitney Bldg., ALR

WALKER, C. C.....Woodward at Warren, Op.

ESCANABA

BOYCE, WILLIAM B.....1019 Ludington St., OALR

GRAND RAPIDS

GRANT, R. T.....Widdicomb Bldg., OALR
 HUIZINGA, J. G.....Widdicomb Bldg., OALR
 ROBERTSON, F. D.....Ashton Bldg., OALR
 ROLLER, L. A.....122 Monroe St., OALR
 WELSH, D. E.....Powers Theater Bldg., OALR

IONIA

SEDGWICK, O. W.....OALR

IRON MOUNTAIN

BOYCE, G. H.....408 E. D. St., OALR

JACKSON

WINTER, G. E.....44 Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B....Kalamazoo National Bank Bldg., OALR
 GRANT, F. E.....Kalamazoo National Bank Bldg., OALR
 WILBUR, E. P.....Kalamazoo National Bank Bldg., OALR

MENOMINEE

ELWOOD, CALVIN R.....927 Main St., OALR

SAGINAW

ROGERS, A. S.....105 S. Jefferson Ave., OALR

MINNESOTA

DULUTH

BRIGGS, F. W.....Lyceum Bldg., ALR
 COLLINS, HOMER.....New Jersey Bldg., OALR
 TILDERQUIST, D. L.....7 E. Superior St., OALR
 TURNBULL, F. M.....Providence Bldg., OALR

FERGUS FALLS

KITTLESON, THEODORE N.....101 Lincoln Ave., OALR

HIBBING

MORSMAN, L. W.....Congdon Bldg., OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BEAUDOUX, H. A.....La Salle Bldg., Op.A
 BENSON, GEO. E.....Metropolitan Bldg., OALR
 BISHOP, C. W.....La Salle Bldg., OALR
 BROWN, E. J.....Syndicate Bldg., OALR
 CAMP, W. E.....Donaldson Bldg., OALR
 CAMPBELL, ROBT. A.....La Salle Bldg., OALR
 CLARK, H. S.....Syndicate Bldg., OALR
 HANSEN, E. W.....Donaldson Bldg., OALR
 HOWE, A. W.....4404 Pleasant Ave., OALR
 KERRICK, STANLEY E.....620 Syndicate Bldg., OALR
 LEE, J. W.....804 Besse Bldg., OALR
 LEWIS, J. D.....La Salle Bldg., OALR
 LITCHFIELD, J. T.....Phys. and Surgeons' Bldg., OALR
 LOOMIS, E. A.....Donaldson Bldg., OALR
 MACINE, J. S.....Donaldson Bldg., OALR
 MATTHEWS, JUSTUSMetropolitan Bldg., LR
 MORSE, J. A.....Donaldson Bldg., OALR
 MURRAY, WM. R.....Nicollet Clinic, OALR

NEWHART, HORACEDonaldson Bldg., OALR
 OBERG, C. M.....Syndicate Blk., OALR
 PARKER, E. H.....La Salle Bldg., LR
 PATTERSON, W. E.....La Salle Bldg., OALR
 PHELPS, K. A.....La Salle Bldg., OALR
 PRATT, F. J., JR.....Metropolitan Bldg., OALR
 PRATT, J. A.....Metropolitan Bldg., OALR
 REYNOLDS, J. S.....La Salle Bldg., OALR
 SMITH, A. E.....Donaldson Bldg., OALR
 STROUT, E. S.....Donaldson Bldg., OALR
 STROUT, G. E.....Donaldson Bldg., OALR
 WATSON, J. A.....Phys. and Surg. Bldg., OALR
 WOOD, DOUGLAS.....Donaldson Bldg., OALR
 WRIGHT, C. D.....Metropolitan Bldg., Op.A

ROCHESTER

BENEDICT, W. L.....Mayo Clinic, Op.
 HEMPSTEAD, BERT E.....Mayo Clinic, ALR
 LILLIE, H. I.....Mayo Clinic, ALR
 LYONS, H. R.....Mayo Clinic, ALR
 NEW, G. B.....Mayo Clinic, LR
 PRANGEN, A. DE H.....Mayo Clinic, Op.

ST. PAUL

BINGER, H. E.....Lowry Bldg., OALR
 BOECKMAN, EGIL.....Lowry Bldg., OALR
 BRAY, E. R.....934 Ashland Ave., OALR
 BROWN, JOHN C.....Lowry Bldg., OALR
 BURCH, F. E.....Hamm Bldg., A
 CONNOR, CHAS. E.....Lowry Bldg., OALR
 FOGARTY, CHAS. W.....1826 Summit Ave., OALR
 FULTON, JOHN E.....728 Lowry Bldg., OALR
 LARSEN, CARL E.....Lowry Bldg., OALR
 LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
 MALONEY, T. J.....Lowry Bldg., OALR
 NELSON, L. A.....Lowry Arcade, Op.A
 ROTHSCHILD, H. J.....Lowry Bldg., OALR
 SHELLMAN, J. L.....Lowry Bldg., OALR

MISSISSIPPI

MERIDIAN

GUTHRIE, J. M.....Rosenbaum Bldg., OALR

OXFORD

GUYTON, B. S.....Carter Bldg., OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., OALR
 EDWARDS, C. J.....1422 Wash., OALR
 HOWARD, E. F.....First National Bank Bldg., ALR

MISSOURI

AURORA

O'DELL, TIMOTHY.....112 A Madison Ave., OALR

CARTHAGE

POST, WINIFRED B.....1380 S. Main St., OALR
 POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

NOYES, G. L.....311 Hitt St., OALR
 SNEED, C. M.....Guitar Bldg., OALR

JOPLIN

PIFER, J. D.....Frisco Bldg.

KANSAS CITY

ALLEN, CHAS. E.....Waldheim Bldg., OALR
 ALTRINGER, ARTHUR N.....Rialto Bldg., ALR
 BEIL, J. W.....Argyle Bldg., OALR
 BELLWS, GEORGE E.....Rialto Bldg., Op.
 BLAKESLEY, T. S.....Lathrop Bldg., OALR
 BOURBON, OLIVER P.....Lathrop Bldg., OALR
 CURDY, R. J.....Rialto Bldg., Op.
 CURRAN, EDW. J.....Waldheim Bldg., Op.
 DAVIS, HARRY B.....Lathrop Bldg., OALR
 FOSTER, HAL.....Altman Bldg., ALR
 GARRISON, B. E.....Argyle Bldg., OALR
 GOSNEY, C. W.....Lathrop Bldg., OALR
 HALL, EDW. P.....124 Hunter Ave., OALR
 HOWARD, J. W.....Rialto Bldg., OALR
 KEITH, W. E.....Waldheim Bldg., OALR
 KIMBERLIN, J. W.....Rialto Bldg., Op.
 LANSING, J. H.....Lathrop Bldg., ALR
 LEA, J. A.....Rialto Bldg., ALR
 LEONARD, A. C.....Bryant Bldg., OALR
 LICHTENBERG, J. S.....Rialto Bldg., Op.
 LORIE, ALVIN.....Commerce Bldg., ALR
 MAY, JAMES W.....800 Minnesota Ave., Op.
 MILLER, HUGH.....Chambers Bldg., OALR
 MOSS, H. E.....Rialto Bldg., OALR
 MOTT, J. S.....Argyle Bldg., OALR
 MYERS, J. L.....Lathrop Bldg., OALR
 McALESTER, A. W., JR.....Bryant Bldg., OALR
 McCARTY, V. W.....Rialto Bldg., ALR
 PAINTER, A. M.....Sharp Bldg., ALR
 PATTERSON, J. M.....Bryant Bldg., OALR
 REED, W. M.....Rialto Bldg., OALR
 ROBERTS, S. E.....Waldheim Bldg., ALR
 RUSSELL, E. L.....Argyle Bldg., OALR
 SCHUTZ, W. H.....Bryant Bldg., OALR
 SELLERS, L. M.....Argyle Bldg., ALR
 SHOEMAKER, S. A.....U. S. Pub. Ser. Hosp., OALR
 SHUMATE, D. L.....Shukert Bldg., OALR
 THOMASON, H. E.....Rialto Bldg., OALR
 TUREMAN, H. G.....Rialto Bldg., OALR
 YAZEL, H. E.....Grand Ave. Temple, OALR

MOBERLY

DUTTON, C. K.....OALR

NEVADA

McLEMORE, TIPTON.....H. C. Moore Bldg., OALR

ST. JOSEPH

KENNEY, W. L.....Corby Forsee Bldg., OALR

MINTON, W. H.....Bartlett Bldg., OALR

PROUD, W. C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ARBUCKLE, M. F.....Lister Bldg., ALR

BALL, J. MOORES.....4500 Olive St., Op.

BARCK, CARL.....Humboldt Bldg., Op.A

BARDENHEIER, F. G. A.....Frisco Bldg., ALR

BLACK, W. D.....Metropolitan Bldg., OALR

BRYAN, W. M. C.....University Club Bldg., ALR

BURNS, S. S.....Humboldt Bldg., ALR

CHARLES, J. W.....Humboldt Bldg., Op.

DONNELL, NEWMAN R.....Metropolitan Bldg., Op.

DYER, CLYDE P.....Metropolitan Bldg., Op.

EWING, ARTHUR E.....Metropolitan Bldg., Op.

GOLDSTEIN, M. A.....3858 Westminster Pl., ALR

GREEN, JOHN, JR.....Metropolitan Bldg., Op.

GUGGENHEIM, L. K.....Carleton Bldg., ALR

GUNDELACH, C. A.....825 University Club Bldg., ALR

HARDESTY, J. F.....Humboldt Bldg., Op.

HARDY, W. F.....Metropolitan Bldg., Op.

HIGBEE, E. H.....Metropolitan Bldg., Op.

HOORN, G. E.....Univ. Club Bldg., ALR

JACOBS, M. W.....Carleton Bldg., Op.

JENNINGS, J. E.....Carleton Bldg., OALR

LAMB, HARVEY.....Metropolitan Bldg., Op.

LEAVY, C. A.....Metropolitan Bldg., ALR

LOEB, H. W.....Humboldt Bldg., ALR

LUEDDE, W. H.....Metropolitan Bldg., Op.

PFINGSTEN, C. F.....508 N. Grand Ave., ALR

POST, LAWRENCE.....Metropolitan Bldg., Op.

POST, MARTIN H.....Metropolitan Bldg., Op.

REIM, HUGO.....Metropolitan Bldg., Op.

SAUER, W. E.....Humboldt Bldg., ALR

SCHOLZ, R. P.....Metropolitan Bldg., ALR

SCHWARTZ, F. A.....Metropolitan Bldg., Op.

SHAHAN, W. E.....Metropolitan Bldg., Op.

SHOEMAKER, W. A.....Carleton Bldg., Op.

SLUDER, GREENFIELD.....3542 Washington Ave., ALR

TOOKER, C. W.....Carleton Bldg., Op.

WESTLAKE, S. V.....Humboldt Bldg., ALR

WIENER, MEYER.....Carleton Bldg., Op.

WOODRUFF, F. E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, J. G.....Ilgenfritz Bldg., OALR

TITSWORTH, GUY.....111 W. 4th St., Op.

SPRINGFIELD

BAILEY, H.....840 Landers Bldg., OALR
 COFFELT, THEODORE A.....Woodruff Bldg., OALR
 KLINGER, THOS. O.....Landers Bldg., OALR

TRENTON

VAUGHN, G. E.....Trenton Trust Bldg., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....Electric Light Bldg., OALR

BUTTE

DAYTON, G. O.....Lewisohn Bldg., OALR
 DONAVAN, J. A.....Phoenix Bldg., OALR
 MORSE, A. W.....Phoenix Bldg., OALR
 POTTER, PETER.....Granate and Alaska Sts., OALR

GREAT FALLS

COULTER, C. F.....1st Natl. Bank Bldg., OALR

LEWISTOWN

DAVIS, FRANK C.....ALR

ROUNDUP

LEWIS, G. A.....29 Main St., OALR

NEBRASKA

BEATRICE

BAIRD, CHARLES G.....OALR
 TUCKER, J. C.....OALR

COLUMBUS

CAMPBELL, C. H.....Columbus State Bk. Bldg., OALR

HOLDREGE

SHIELDS, W. D.....OALR

KEARNEY

FOX, CHAS. H.....2116 Central Ave., OALR

LINCOLN

BROOKS, E. B.....Terminal Bldg., OALR
 COOK, S. E.....Richards Block, OALR
 CURTIS, W. L.....612 Security Mutual Bank Bldg., OALR
 DAYTON, W. L.....Funke Bldg., OALR
 FURGASON, A. P.....Richards Block, OALR
 HOMPES, J. J.....Security Mutual Bldg., OALR
 SANDERSON, DAVID D.....Funke Bldg., OALR
 WILLIAMS, J. P.....Funke Bldg., OALR
 WOODWARD, J. M.....Richards Block
 ZEMER, S. G.....Security Mutual Bldg., OALR

NORFOLK

GADBOIS, A. E.....421 Norfolk Ave., OALR

OMAHA

BANISTER, J. M.....Brandeis Theater Bldg., OALR
 CALLFAS, W. F.....Brandeis Bldg., ALR
 FAIRCHILD, NORA M.....600 Brandeis Theater Bldg., Op.
 GIFFORD, HAROLD.....Brandeis Bldg., Op.A
 GIFFORD, SANFORD R.....702 Brandeis Theater Bldg., Op.
 HANEY, W. P.....Brandeis Theater Bldg., OALR
 ISAAC, DAVID.....Brandeis Theater Bldg., OALR

KNOVDE, A. R.....Omaha National Bank Bldg., ALR
 KULLY, B. M.....631 City Natl. Bk. Bldg., ALR
 LEMERE, H. B.....Brandeis Bldg., OALR
 OWEN, F. S.....Brandeis Bldg., OALR
 PATTON, J. M.....Brandeis Bldg., OALR
 POTTER, GEORGE B.....402 City Natl. Bk. Bldg., ALR
 POTTS, J. B.....Brandeis Bldg., AL
 RUBENTHAL, CLARENCE.....Brandeis Bldg., OALR
 TUCKER, J. C.....Brandeis Bldg., OALR
 UREN, C. T.....Omaha National Bank Bldg., OALR
 WHERRY, W. P.....Brandeis Theater Bldg., OALR

NEW HAMPSHIRE

HANOVER

CARLETON, E. H.....ALR

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. C.....124 S. Illinois St., ALR

McGIVERN, CHAS. S.....805 Pacific Ave., ALR

STICKNEY, O. D.....92 Pacific Ave., ALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

JERSEY CITY

CHAMBERS, T. R.....15 Exchange Pl., OALR

PYLE, WALLACE.....18th and Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CHATTIN, J. F.....671 Broad St., Op.

HURF, J. W.....71 Washington St., OALR

O'CONNOR, F. O.....671 Broad St., OALR

ORTON, H. B.....671 Broad St., ALR

QUINBY, W. O'G.....14 James St., OALR

SHERMAN, E. S.....Wiss Bldg., OALR

ZEHNDER, C. A.....180 Fairmount Ave., OALR

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

TRENTON

ADAMS, CHARLES.....52 W. State St., OALR

CRANE, J. WELLINGTON.....128 Third St., ALR

WEST HOBOKEN

SACCO, A. G.....440 Clinton Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L.....521 Luna Blvd., OALR

NEW YORK

ALBANY

BEDELL, A. J.....	344 State St., Op.A
BEGGS, W. F.....	2 Lombardy St., OALR
DOWLING, J. I.....	116 Washington Ave., OALR
JUDGE, H. V.....	228 State St., Op.

BINGHAMPTON

WATSON, H. D.....	151 Front St., OALR
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BROOKLYN

ARROWSMITH, HERBERT.....	170 Clinton St., ALR
HANCOCK, J. C.....	135 Cambridge Pl., Op.
MCCLELLAND, L. A.....	78 McDonough St., ALR

BUFFALO

ANDREWS, H. D.....	23 Allen St., Op.
BENNETT, A. G.....	26 Allen St., Op.
BEYER, LOUIS J.....	449 Delaware Ave., ALR
BLAAUW, E. E.....	1901 Ashland Ave., OALR
BROWN, C. M.....	510 Delaware Ave., ALR
COTT, C. C.....	483 Delaware Ave., ALR
COWPER, H. W.....	543 Franklin St., Op.
EDSON, RAY.....	560 Delaware Ave., Op.
FAIRBAIRN, JOHN.....	503 Delaware Ave., ALR
FLAGG, J. D.....	473 Virginia Ave., Op.A
FRANCIS, L. M.....	636 Delaware Ave., Op.
GLOSSER, HERBERT H.....	448 Franklin St., OALR
HEALEY, J. F.....	503 Delaware Ave., ALR
HOWE, LUCIEN.....	520 Delaware Ave., Op.
HUBBARD, A. E.....	372 Franklin St., Op.
LEWIS, F. P.....	454 Franklin St., Op.
MARCH, CLARA A.....	465 Ashland Ave., Op.
PHILLIPS, W. L.....	469 Franklin St., Op.
RENNER, W. S.....	341 Linwood Ave., ALR
SATTERLEE, R. H.....	187 Delaware Ave., Op.
SERNOFFSKY, I.....	37 Allen St., Op.
STARR, E. G.....	523 Delaware Ave., Op.
WEED, HARRY M.....	196 Lincoln Ave., Op.

DUNKIRK

JACKLE, A. F.	63 E. 4th St., Op.
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GENEVA

SPENGLER, J. A.....	423 Main St., OALR
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ITHACA

BULL, E. L.....	124 E. State St., OALR
KIRKENDELL, J. S.....	315 N. Aurora, OALR
WILSON, R. C.....	208 E. State St., OALR

JAMESTOWN

HOTCHKISS, W. W.....	195 Forest Ave., OALR
REGER, H. S.....	New Wellman Bldg., OALR

KENMORE

WURTZ, W. J. M.....	2808 Delaware Ave., ALR
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LIBERTY

DWORETSKY, JULIUS.....7 Law St., ALR

LOCKPORT

RINGUEBERG, EUGENE.....15 Main St., Op.

MT. VERNON

THOMSON, J. J.....3 Park Ave., OALR

NEW YORK CITY

AUERBACH, JULIUS.....120 W. 86th St., ALR
 BALLIN, M. J.....108 E. 60th St., ALR
 BELL, GEO. HUSTON.....40 E. 41st St., Op.
 BERENS, CONRAD, JR.....9 E. 46th St., Op.
 BRUDER, JOSEPH.....230 W. 79th St., OALR
 CARTER, W. W.....2 W. 67th St., ALR
 COHEN, MARTIN.....1 W. 85th St., OALR
 COHN, FELIX.....31 E. 60th St., ALR
 DAVIS, A. E.....47 E. 57th St., Op.
 DAVIS, G. E.....42 W. 77th St., OALR
 DIXON, G. S.....40 E. 41st St., R
 DUANE, ALEXANDER.....139 E. 37th St., Op.
 FORBES, H. H.....40 E. 41st St., ALR
 FREUDENTHAL, WOLFF.....24 W. 88th St., ALR
 FRIDENBERG, P. H.....38 W. 59th St., OALR
 FRIEDMAN, DAVID.....44 W. 77th St., ALR
 GATEWOOD, W. L.....15 E. 48th St., ALR
 GLOGAU, OTTO.....64 E. 91st St., OALR
 GOTTLIEB, M. J.....814 West End Ave., ALR
 GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
 GUTTMAN, J.....616 Madison Ave., OALR
 HASKIN, W. H.....40 E. 41st St., ALR
 HAYS, H. N.....2178 Broadway ALR
 HOPKINS, W. E.....515 Park Ave., OALR
 HURD, L. M.....39 E. 50th St., ALR
 IMPERATORI, C. J.....17 E. 38th St., ALR
 INGERMAN, SERGIUS.....Hotel Brunswick, OALR
 IRWIN, F. N.....114 E. 54th St., OALR
 JACOBS, S. M.....375 E. 149th St., OALR
 JARECKY, HERMAN.....138 W. 86th St., OALR
 JUDD, H. B.....47 E. 57th St., ALR
 KERRISON, P. D.....58 W. 56th St., ALR
 KEY, B. W.....7 W. 49th St., Op.
 KING, J. J.....40 E. 41st St., ALR
 KNAPP, ARNOLD.....10 E. 54th St., Op.
 KOPETZKY, S. J.....51 W. 73rd St., ALR
 KRUG, E. F.....12 W. 44th St., OALR
 LA VIGNE, A. A.....11 E. 48th St., OALR
 LUBMAN, MAX.....616 Madison Ave., ALR
 MAY, C. H.....698 Madison Ave., Op.
 MAYBAUM, J. E.....17 E. 38th St., ALR
 MAYER, EMIL.....40 E. 41st St., ALR
 MCCOY, JOHN.....157 W. 73d St., ALR
 MEIERHOF, E. L.....1140 Madison Ave., OALR

MITTENDORF, A. D.....	399 Park Ave., Op.
MITTENDORF, W. K.....	115 E. 53rd St., Op.A
MORTIMER, W. GOLDEN.....	58 Central Park, W., OALR
NISSSELSON, MAX.....	616 Madison Ave., OALR
OPPENHEIMER, SEYMOUR.....	45 E. 60th St., OALR
PAYNE, S. M.....	542 Fifth Ave., OALR
PHILLIPS, W. C.....	40 W. 47th St., ALR
REESE, R. G.....	50 W. 52nd St., Op.
SCHILLER, A. N.....	225 West End Ave., ALR
STROUSE, A. N.....	153 W. 79th St., OALR
STURGES, L. F.....	128 W. 87th St., LR
VOORHEES, IRVING W.....	14 Central Park, W., ALR
WEEKS, JOHN E.....	46 E. 57th St., Op.
WEINSTEIN, JOSEPH.....	261 Central Park, W., ALR
WHEELER, J. M.....	30 W. 59th St., Op.
WIENER, ALFRED.....	550 Park Ave., OALR
YANKAUER, SIDNEY.....	616 Madison Ave., ALR

NIAGARA FALLS

PRICE, N. W.....	445 Third St., OALR
ROOKER, A. L.....	1347 Michigan Ave., ALR

OLEAN

TINDOLPH, LEA W.....	First Natl. Bk. Bldg., OALR
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ONEIDA

CROCKETT, R. L.....	Op.
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POUGHKEEPSIE

KRIEGER, W. A.....	36 Market St., OALR
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ROCHESTER

BARBER, FRANK.....	259 Alexander St., Op.
CARROLL, G. G.....	614 Main St., W., OALR
CLARK, L. H.....	337 Monroe Ave., OALR
MCDOWELL, N. D.....	275 Alexander St., OALR
MORRIS, A. G.....	41 Gibbs St., OALR
SHAPERO, I. M.....	365 East Ave., OALR

SYRACUSE

BROWN, M. G.....	802 University Bldg., ALR
BRITEN, G. S.....	University Blk., OALR
BRUST, H. O.....	720 S. Crouse Ave., ALR
FOWLER, S. R.....	University Bldg., ALR
KLINE, H. G.....	University Blk., ALR

TROY

SULZMAN, F. M.....	1831 Fifth Ave., OALR
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UTICA

BEATTIE, W. HENRY.....	252 Genesee St., OALR
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NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....	73 Haywood St., OALR
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CHARLOTTE

MATHESON, J. P.....	511 Independence Bldg., OALR
PEELER, C. N.....	W. 7th St., OALR

SLOAN, HENRY L.....Independence Bldg., OALR
WHISNANT, A. M.....Realty Bldg., OALR

GREENSBORO

REAVES, W. P.....117 W. Sycamore St., OALR

NEW BERN

DANIELS, R. L.....OALR

RALEIGH

WRIGHT, J. B.....Citizens Natl. Bk. Bldg., OALR

SALISBURY

BRAWLEY, R. V.....Wallace Bldg., OALR

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

NORTH DAKOTA

FARGO

RINDLAUB, ELIZABETH P.....De Lendrecie Blk., OALR

RINDLAUB, J. H.....De Lendrecie Blk., OALR

RINDLAUB, M. P., JR.....De Lendrecie Blk., OALR

MINOT

McCONNELL, A. D.....Colcord Bldg., OALR

WAHPETON

LANCASTER, WILSON.....310 Dakota Ave., OALR

RICE, C. P.....Sterns Bldg., OALR

OHIO

AKRON

BROWN, L. E.....2nd Natl. Bank Bldg., OALR

MOORE, T. K.....Hamilton Bldg., OALR

STEVENSON, D. W.....165 E. Market St., OALR

ALLIANCE

KING, G. L.....537 E. Market St., OAR

BELLEFONTAINE

BUTLER, ROBERT H.....227 W. Columbus Ave., OALR

HARBERT, J. P.....Canby Blk., OALR

CANTON

SCHILD, E. H.....9th and Cleveland, Op.A

CINCINNATI

ALLEN, S. E.....22 W. 7th St., ALR

AUFMWASSER, H.....OALR

AYRES, W. McL.....4 W. 7th St., Op.

HANS, C. L.....2900 Grasselli Ave., OALR

HARRIS, W. C.....Groton Bldg., ALR

HEFLEBOWER, R. C.....The Lancaster, OALR

HINNEN, G. A.....1343 Delta Ave., OALR

IGLAUER, SAMUEL.....Livingston Bldg., ALR

KING, C.....Union Central Bldg., Op.

KING, EDWARD.....605 Traction Bldg., ALR

LAMB, F. W.....7th and Vine Sts., OALR

MITHOEFER, WILLIAM.....19 W. 7th St., ALR

MURPHY, J. W.....Union Central Life Bldg., OALR

MURPHY, W. E.....7th and Race Sts., OALR

PHINNEY, F. D.....22 W. 7th St., OALR
 RAY, VICTOR19 W. 7th St., OALR
 SIEGEL, F. X.....2700 Union Central Bldg., OALR
 STANBERRY, HENRYProvident Bk. Bldg., Op.AR
 STEVENSON, ROBERT22 W. 7th St., ALR
 STEWART, T. M.....Union Trust Bldg., OALR
 STRICKER, LOUISGroton Bldg., Op.
 TANGEMAN, H. F.....Central Bldg., ALR
 THOMSON, E. H.....13 W. 7th St., OALR
 URNER, M. H.....Union Central Life Bldg., OALR
 VAIL, D. T.....24 E. 8th St., Op.

CLEVELAND

ABBOTT, W. J.....628 Union Bldg., OALR
 BRUNER, W. E.....Guardian Bldg., Op.
 BURKE, T. A.....Rose Bldg., OALR
 COGAN, J. E.....Rose Bldg., OALR
 CUTLER, FRANKLIN E.....1025 Schofield Bldg., ALR
 FORSYTHE, S. T.....1011 Euclid Ave., OALR
 INGERSOLL, J. M.....Osborn Bldg., ALR
 KENDALL, M. R.....Osborn Bldg., ALR
 LARGE, S. H.....Rose Bldg., ALR
 LENKER, J. N.....Osborn Bldg., ALR
 METZ, R. B.....Guardian Bldg., Op.
 METZENBAUM, MYRONRose Bldg., ALR
 MONSON, S. H.....Ainsfield Bldg., OALR
 McDONALD, C. L.....Ainsfield Bldg., OALR
 NELSON, C. F.....Schofield Bldg., OALR
 PITKIN, C. E.....Osborn Bldg., ALR
 PRENDERGAST, D.1110 Euclid Ave., OALR
 QUITTNER, S. S.....3912 Prospect Ave., OALR
 RUST, E. G.....Hanna Bldg., Op.A
 SHACKELTON, W. E.....Osborn Bldg., Op.
 SHIRAS, H. H.....Guardian Bldg., Op.
 SIMONDS, O. F.....Rose Bldg., Op.
 STOTTER, A. L.....1148 Euclid Ave., OALR
 STOTTER, JAMES1148 Euclid Ave., OALR
 TRIPP, I. C.....Rose Bldg., OALR
 TUCKERMAN, W. C.....Osborn Bldg., Op.
 TUCKERMAN, W. H.....Osborn Bldg., OALR
 WAUGH, J. M.....3095 Fairfax, ALR
 WOLFENSTEIN, LEORose Bldg., Op.

COLUMBUS

BEATTY, H. G.....150 E. Broad St., OALR
 BROWN, J. E.....239 E. Town St., OALR
 CLARK, I. G.....188 E. State St., OALR
 HAUER, A. M.....327 E. State St., OALR
 MEANS, C. S.....131 E. State St., OALR
 PROUT, A. W.....370 E. Town St., OALR
 SANOR, D. G.....206 E. State St., OALR
 SCHAEFFER, G. C.....216 E. State St., OALR
 SILBERNAGEL, C. E.....283 E. State St., OALR

SULZER, G. A.....200 E. State St., OALR
 THOMAS, F. W.....74 S. 5th St., OALR
 TIMBERMAN, ANDREW.....112 E. Broad St., OALR
 WOLFE, A. C.....350 E. State St., ALR
 WRIGHT, J. W.....Central National Bank Bldg., Op.

DAYTON

BINKLEY, R. S.....Fidelity Med. Bldg., OALR
 BLACKBURN, W. J.....Reibold Bldg., OALR
 BONNER, HORACE.....Fidelity Med. Bldg., OALR
 DUTROW, H. V.....1040 Fidelity Medical Bldg., OALR
 FARMER, A. G.....Fidelity Med. Bldg., OALR
 FOUTS, JOHN D.....Fidelity Medical Bldg., OALR
 HARRIS, H. B.....Fidelity Med. Bldg., OALR
 MILLETTE, J. W.....Reibold Bldg., OALR
 WEBSTER, R. M.....Reibold Bldg., OALR

ELYRIA

GILL, GEORGE146 Middle Ave., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts.

LIMA

STUEBER, F. G.....Main and North Sts., OALR
 STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....424 Broadway, OALR

MANSFIELD

GARBER, J. M.....48 Park Ave., OALR
 WISE, R. C.....News Bldg., OALR

MARIETTA

SAUER, W. W.....Box 416, OALR

MARION

BRICKLEY, D. W.....196 W. Center St., OALR

MIDDLETOWN

WILLIAMS, WALTER H..... OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SALEM

HILL, A. J.....301 Citizens Natl. Bk. Bldg., OALR

SANDUSKY

BLISS, C. B.....411 Columbus Ave., OALR

SPRINGFIELD

EASTON, J. C.....Fairbanks Bldg., OALR
 HARTLEY, F. A.....Fairbanks Bldg., OALR
 HOGUE, D. W.....Fairbanks Bldg., OALR
 MINOR, C. L.....Fairbanks Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg.

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDERDYCE, W. W.....Nasby Bldg., OALR
 ALTER, F. W.....Colton Bldg., OALR
 DENMAN, IRA O.....Ohio Bldg., OALR
 HOBART, BERTHA K.....Produce Exchange Bldg., OALR

JACOBI, FRANK Colton Bldg., OALR
 KELLER, T. F. The Spitzer, OALR
 KING, CHARLES R. Spitzer Bldg., OALR
 LUKENS, CHARLES 218 Michigan Ave., OALR
 SNYDER, W. H. 211 Ontario St., OALR
 STEINFELD, A. L. 234 Michigan St., OALR

XENIA

MADDEN, REED. Allen Bldg., OALR
 SHIELDS, L. Allen Bldg., ALR

YOUNGSTOWN

GIBSON, R. D. Dollar Savings and Trust Bldg., OALR
 HARTZELL, S. M. Dollar Bank Bldg., OALR

ZANESVILLE

BARON, F. S. Peoples Bank Bldg., OALR
 CULBERTSON, L. R. Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D. Central Natl. Bk. Bldg., OALR

GUTHRIE

BARKER, CHAS. B. 224½ W. Oklahoma Ave., OALR

MCALISTER

DAVIS, J. E. Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M. Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E. 706 1st Natl. Bank Bldg., OALR
 FERGUSON, E. I. 607 1st Natl. Bank Bldg., OALR
 GUTHRIE, A. L. Amer. Natl. Bank Bldg., OALR
 MCHENRY, D. D. Colcord Bldg., OALR
 NEWTON, L. A. Colcord Bldg., OALR
 TODD, H. C. Colcord Bldg., OALR

TULSA

BRASWELL, C. J. 726 Mayo Bldg., OALR
 ROTH, A. W. Palace Bldg., OALR

OREGON

LA GRANDE

BOUVY, HARRY. New Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE Oregonian Bldg., OALR
 BRUERE, G. E. Journal Bldg., OALR
 DAVIS, RALPH F. Selling Bldg., OALR
 FENTON, RALPH A. 616 Journal Bldg., OALR
 HENTON, GEORGE EARL. Morgan Bldg., OALR
 JOHNSTON, WILSON Stevens Bldg., OALR
 KIEHLE, F. A. Corbett Bldg., OALR
 KISTNER, F. B. Stevens Bldg., ALR
 MCCOOL, JOSEPH L. Stevens Bldg., Op.

PENNSYLVANIA

ALTOONA

GLOVER, S. P. 1118 12th Ave., OALR

BEAVER

SCROGGS, J. J.....218 E. 3rd St., OALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....I. O. O. F. Temple, OALR

HAZLETT, L. R.....117 S. Main St., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR

STAHLMAN, F. C.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.

ERIE

DENNIS, D. N.....221 W. 9th St., Op.A

DENNIS, E. P.....311 W. 10th St., OALR

DUNN, I. J.....Masonic Temple, OALR

FUST, J. H. E.....234 W. 6th St., OALR

HEARD, C. F.....Masonic Temple, OALR

JACKSON, J. D.....2324 W. 8th St.

RUSSELL, J. A.....206 Masonic Temple, ALR

SCHLINDWEIN, G. W.....138 W. 9th St., OALR

SHREVE, O. M.....162 W. 8th St., OALR

WRIGHT, KATHERINE L.....247 W. 8th St., ALR

FRANKLIN

JOBSON, G. B.....Printz Bldg., OALR

GREENSBURG

McKEE, C. W.....Coulter Bldg., OALR

HARRISBURG

DAILEY, G. L.....713 N. 3rd St., OALR

FARNSLER, H. H.....1438 Market St., OALR

PARK, J. W.....32 N. 2d St., OALR

REBUCK, C. S.....412 N. 3d St., OALR

HAZELTON

REICHE, O. C.....328 W. Broad St., OALR

HUNTINGDON

SEARS, W. H.....514 Penn St., OALR

JOHNSTOWN

BARKER, O. G. A.....Johnstown Trust Bldg., Op.

HARRIS, CLARENCE M.....Johnstown Trust Bldg., OALR

MIDDLETOWN

GEORGE, H. W.....19 N. Union St., Op.

NEW BRIGHTON

NORTON, ROY R.....Masonic Bldg., OALR

NEW KENSINGTON

McCONNELL, T. E.....Alter Bldg., OALR

STEIM, JOSEPH M.....1st Natl. Bk. Bldg., Op.

PHILADELPHIA

ALBRIGHT, P. M.....League Island Naval Hosp., ALR

APPLEMAN, L. F.....308 S. 16th St., Op.

BALDWIN, KATE W.....1117 Spruce St., ALR

BALENTINE, P. L.....1524 Chestnut St., Op.

BOEHRINGER, H. W.....1811 S. 22nd St., Op.

BRINKERHOFF, NELSON M.....	1831 Chestnut St., Op.
BROWN, S. H.....	1901 Mt. Vernon St., Op.
BRUMM, SETH A.....	Stock Exchange Bldg., ALR
BUTLER, MARGARET F.....	1831 Chestnut St., ALR
BUTT, M. M.....	2045 Chestnut St., Op.
COATES, G. M.....	1736 Pine St., ALR
COHEN, SAMUEL.....	1626 Spruce St., ALR
CRAMPTON, G. S.....	1700 Walnut St., Op.
CREIGHTON, W. J.....	1905 Chestnut St., Op.
CROSKEY, J. W.....	1909 Chestnut St., OALR
DAVIS, J. L.....	135 S. 18th St., ALR
DEICHLER, LYN WALTER.....	2028 Chestnut St., OALR
DINTENFASS, HENRY.....	1714 Pine St., ALR
EARL, HARRY D.....	3717 Spruce St., ALR
ERSNER, M. S.....	1729 Pine St., ALR
FELT, C. L.....	2007 Chestnut St., ALR
FOX, L. W.	303 S. 17th St., Op.
GLEASON, E. B.....	2033 Chestnut St., ALR
GODDARD, H. M.....	1531 Spruce St., ALR
GRISCOM, J. M.....	1925 Chestnut St., Op.
HAWMAN, E. G.....	4051 Broad St., OALR
HEED, C. R.....	1205 Spruce St., Op.
HERBERT, A. W.....	119 E. Lehigh Ave., OALR
HOLLOWAY, T. B.....	1819 Chestnut St., Op.
HUSIK, DAVID N.....	1303 Locust St., ALR
JACKSON, C.....	128 S. 10th St., L
KAUFMAN, A. S.....	1923 Spruce St., ALR
KEELER, J. C.....	254 S. 16th St., ALR
KRAUSS, FREDERICK.....	1701 Chestnut St., OALR
LAESSLE, H. A.....	The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....	Medical Arts Bldg., Op.
LEWIS, FIELDING O.....	259 S. 17th St., ALR
LOFTUS, J. E.....	605 Medical Arts Bldg., ALR
LOVE, LOUIS F.....	1305 Locust St., Op.
LUKENS, R. McD.....	1308 Hunting Park Ave., ALR
MACFARLIN, DOUGLAS	1805 Chestnut St., OALR
MACKENZIE, ALICE V.....	1724 Spruce St., OALR
MACKENZIE, GEORGE W.....	1724 Spruce St., ALR
MILLER, E. B.....	2008 Walnut St., Op.
MITCHELL, E. K.....	704 Lehigh Ave., ALR
MOORE, W. F.....	255 S. 16th St., ALR
O'REILLY, C. A.....	1901 Chestnut St., ALR
PETER, LUTHER C.....	1529 Spruce St., Op.
PONTIUS, P. J.....	1831 Chestnut St., OALR
POSEY, WM. CAMPBELL.....	21st and Chestnut Sts., Op.
RADCLIFFE, MCCLUNEY.....	1906 Chestnut St., Op.
REESE, WARREN S.....	230 S. 21st St., Op.
RIDPATH, R. F.....	1928 Chestnut St., ALR
ROMMEL, J. C.....	4501 N. Broad St., OALR
SARTAIN, PAUL J.....	2006 Walnut St., Op.
SCARLETT, HUNTER W.....	2228 Locust St., Op.
SCHATZ, H. A.....	2035 Chestnut St., ALR

SCHNEIDEMAN, T. B.....Professional Bldg., Op.
 SCHWEINITZ, G. E. DE.....1705 Walnut St., Op.
 SCHWENK, P. N. K.....1417 N. Broad St., Op.
 SENER, W. J.....130 S. 18th St., OALR
 SHEMELEY, W. G., JR.....1724 Spruce St., OALR
 SKILLERN, R. H.....1928 Chestnut St., LR
 SMITH, S. McC.....1420 Spruce St., ALR
 SPENCER, W.....1830 S. Rittenhouse Sq., OALR
 STILES, C. M.....1831 Chestnut St., Op.
 STOUT, PHILIP S.....Medical Arts Bldg., ALR
 STROUSE, FRED M.....1301 Spruce St., ALR
 SWEET, W. M.....1205 Spruce St., Op.
 WAGERS, A. J.....The Lenox, 13th and Spruce Sts., ALR
 WELCH, R. S. G.....League Island Nav. Hosp., OALR
 WILLIAMS, H. J.....5908 Green St., Germantown, ALR
 ZEIGLER, S. L.....1625 Walnut St., Op.
 ZENTMAYER, WILLIAM.....1505 Spruce St., Op.

PITTSBURGH

BEANE, GEORGE W.....Jenkins Arcade Bldg., OALR
 BERNATZ, C. F.....Park Bldg., Op.
 BLAIR, W. W.....Diamond Bank Bldg., Op.
 BROWN, W. E.....Union Arcade Bldg., OALR
 CARSON, W. E.....Jenkins Arcade Bldg., Op.
 CRAIGHEAD, NANCY B.....Jenkins Arcade Bldg.
 CURRY, G. E.....Westinghouse Bldg., Op.
 DAY, EWING W.....Westinghouse Bldg., ALR
 DICKINSON, B. M.....Union Arcade Bldg., ALR
 FRANK, A. C.....Diamond Bank Bldg., ALR
 GROSS, JULIUS E.....Jenkins Arcade Bldg., OALR
 GUERINOT, A. J.....Jenkins Arcade Bldg., OALR
 HECKEL, E. B.....Jenkins Arcade Bldg., Op.
 KREBS, A.Jenkins Arcade Bldg., Op.
 LINN, J. G.....Highland Bldg., OALR
 MARKEL, J. C.....Westinghouse Bldg., Op.
 MARSHAL, WATSONDiamond Bank Bldg., ALR
 McCREADY, J. H.....Empire Bldg., ALR
 McKEE, GEORGE J.....1118 Westinghouse Bldg., ALR
 MACLACHLAN, A. A.....Empire Bldg., ALR
 MEANOR, W. C.....Westinghouse Bldg.
 MURDOCH, J. F.....Bessener Bldg., Op.
 PATTERSON, E. J.....Westinghouse Bldg., ALR
 SEEGBMAN, SIMONJenkins Arcade Bldg., ALR
 SHUMAN, G. H.....Park Bldg., Op.
 SIMPSON, JOHN R.....1005 Westinghouse Bldg., ALR
 SMITH, S. S.....Jenkins Arcade Bldg., Op.
 STIEREN, EDWARDUnion Arcade Bldg., Op.
 STIMSON, GEO. W.....Jenkins Arcade Bldg., ALR
 STURM, S. A.....Jenkins Arcade Bldg., Op.
 TAYLOR, MARTIN C.....415 Warrington Ave.
 TODD, G. C.....307 Westinghouse Bldg., ALR
 TURNER, HUNTER H.....Jenkins Arcade Bldg., Op.
 VAN KIRK, V. E.....Union Arcade Bldg., Op.

WEIMER, EDGAR S.....332 Lehigh Ave., Op.
 WEISSER, E. A.....Empire Bldg., Op.
 WIBLE, E. E.....Diamond Bank Bldg., Op.
 WISHART, C. A.....Jenkins Arcade Bldg., Op.

READING

STOCKLER, J. A.....332 N. 5th St., OALR

RIDGEWAY

McALISTER, J. C.....10 S. Mill Ave., OALR

SCHUYLKILL HAVEN

MOORE, GEORGE H.....200 Main St., OALR

SCRANTON

CORSER, J. B.....Scranton Private Hospital, OALR

FREY, C. L.....529 Pine St., OALR

SUNBURY

BECKER, H. M.....49 S. 4th St., OALR

UPPER DARBY

SHANNON, C. E.,.....104 Copley Rd., Op.

WARREN

BALL, M. V.....214 Penn Ave., W., OALR

WASHINGTON

McKENNAN, J. W.....Washington Trust Bldg., Op.

McMURRAY, J. B.....Washington Trust Bldg., OALR

WILKES-BARRE

BUCKMAN, E. V.....70 S. Franklin St., OALR

CARR, G. W.....30 S. Franklin St., OALR

KISTLER, O. F.....43 N. Franklin St., Op.A

McLAUGHLIN, P. A.....73 S. Washington St., OALR

SCHAFFERT, N. L.....57 S. Washington St., OALR

TAYLOR, L. H.....83 S. Franklin St., OALR

YORK

KLINEDIENST, J. F.....220 S. George St., OALR

RHODE ISLAND

NEWPORT

JACOBY, D. P. A.....106 Touro, Op.AL

PROVIDENCE

BIGELOW, I. N.....124 Waterman St., ALR

CAPRON, F. P.....118 Angell St., OALR

HARVEY, N. D.....114 Waterman Ave., OALR

LEECH, J. W.....111 Broad St., OALR

MESSINGER, H. C.....170 Broad St., Op.

MUNCY, W. M.....25 Waterman St., OALR

PORTER, L. B.....117 Waterman St., OALR

SOUTH CAROLINA

CHARLESTON

KOLLOCK, C. W.....86 Wentworth, OALR

COLUMBIA

WHALEY, E. M.....1430 Blanding St., OALR

GREENVILLE

CARPENTER, E. W.....201 E. North St., ALR

MAULDIN, L. O.....Wallace Bldg., OALR

ROCK HILL

TWITTY, W. E.....Peoples Natl. Bk. Bldg., OALR

SOUTH DAKOTA

ABERDEEN

ALWAY, ROBERT D.....423 S. Lincoln St., OALR

HURON

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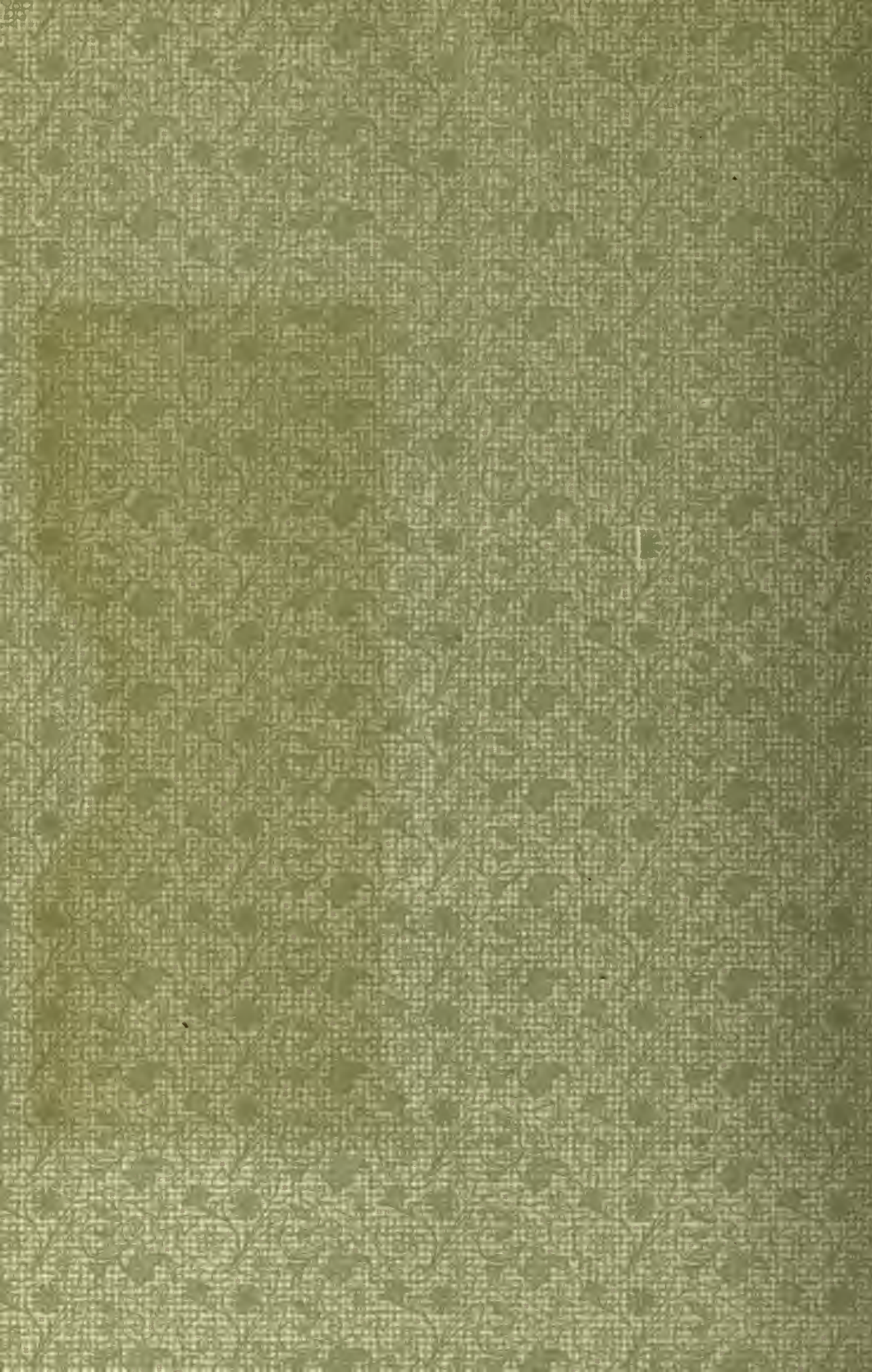
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